# THE HEART AS A POWER-CHAMBER

HARRINGTON SAINSBURY

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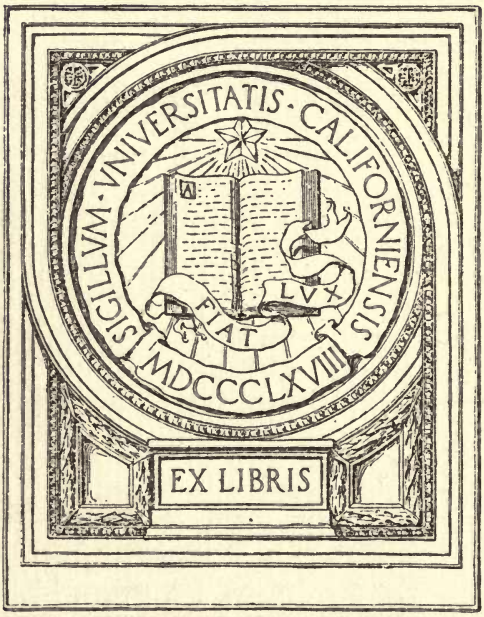

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*Oxford Medical Publications*

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## A CONTRIBUTION TO CARDIO-DYNAMICS

BY

HARRINGTON SAINSBURY

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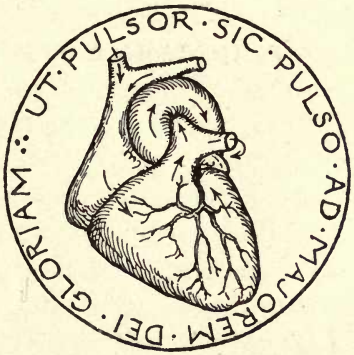


Hanc mirabilem structuram
BORELLI: *de Motu Animalium*

And what shoulder, and what art
Could twist the sinews of thy heart?
WILLIAM BLAKE

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# PREFACE

THE following pages present an endeavour to bring together the facts of anatomy, as they show themselves in the dissecting room and the post-mortem room, along with the observations of physiology, as, in the laboratory, these disclose the living principle, the Archæus, working within the organs and tissues.

In the dissecting room, as in the post-mortem room, we see structure designed to contain and control and give effect to certain forces, and to the manifestations which such forces and structures together generate we apply the term "vital." In the absence of structure force has no direction; it is disembodied, so to speak; it is formless. In the absence of force, structure is incapable of expression; it is powerless; and it is thus that we see the organs and tissues after death, and for this reason that we miss the true significance of structure, which to be interpreted fully must be seen in action. What is to be done to make more profitable our exploration of the lifeless organs and tissues? for to the end of time we shall continue to peer and pry, with scalpel and forceps and microscope, notwithstanding the absence of the vital principle.

Years ago, in some lecture or address, I forget the occasion and the reference, Sir Samuel Wilks quoted from the New Testament the memorable words, *τί ζητεῖτε τὸν ζῶντα μετὰ τῶν νεκρῶν* (Why seek ye the living among the

dead ?)—forbidding, I think, or, if not forbidding, discouraging the search. Well, it is to that very quest that I would invite, for though it be true that the body is tenantless at the time of our examination, a close inspection of the empty chambers and passages, even to the crannies, will discover signs which tell of the departed guest, its ways and doings, its comings and goings, the while it frequented the body. Thus a thickening and opacity here may indicate the repeated impact of some force or forces; a dilatation there the effect of distending activities; whilst in another part the fusion of the segments of a valve may signify, now an obstruction to flow in one direction, and now an incompetence to stem flow in an opposite direction, according as the fused valve comes into action at different phases of the cardiac cycle; and so on. This it is then that we must look for, whether as anatomist or pathologist, viz. the evidence of past activities in the structures which death has brought to a standstill. This will enable us to conjure back vitality and see it in possession, moving the whole organism, in all its parts, to a designed and co-operant end. Thus we shall visualize the organs and tissues dynamically, and to the extent to which we succeed in so doing our anatomy and pathology will become correspondingly living.

To the thoughtful student of Anatomy and Pathology the most salient features in his studies will be the design and foresight which these reveal: design, to the compassing of a given objective; foresight, to the securing that attainment shall not be frustrated by what one may term the accidentals of life. This latter, this

prevision and provision, is so striking that it has been regarded as a living, protecting entity and has been named the "vis medicatrix naturæ." It deserves its name; for though it has its limitations and on occasion may work to the detriment of the body as a whole, even whilst safeguarding the part, yet in the general its tendency to re-establish the disturbed health is so strong, that we dare never have it out of mind and must always endeavour to co-operate with it. The *dictum primum non nocere* envisages this very force, bidding us see to it that we do not run counter to the natural rehabilitating powers of the tissues.

And what are we to say of design, that other salient feature which the study of structure brings out? This at least, that the more we look at it and into it, the more does its supreme art appear, and nowhere more so than when we search the chambers of the heart. Well may Borelli call this organ "*hanc mirabilem structuram*," and well may William Blake, the mystic, innocent of science and all its ways, exclaim in that strange poem, "The Tiger":

> "And what shoulder, and what art
> Could twist the sinews of thy heart?"

Whence came the inspiration which thus spoke, and whence the insight? Did he happen to know the course of the fibres of the heart as they radiate from the base and stream towards the apex, there turning inwards in a vortex, or twist, to be reflected upwards along the inner surface, and finally brought back to the base. It is almost certain that he did not, though by the labours of Borelli and of Lower, and other workers in the

seventeenth century, the knowledge was there. But if he had known it all he could not have expressed in nobler words our wonder and admiration at this marvel of intricate design. From out this coil of twisted sinews there issues the magnificent sequence of heart-beats which measures out our length of days : can one wonder at the enthusiasm of those earlier investigators who first caught a glimpse of the order which reigned within this intricacy, and of the fullness of purpose which it disclosed ?

St. Augustine of Hippo tells us in his *Confessions* that as a young man, when the love of art and the search after beauty absorbed him to the exclusion of almost everything else, he wrote " two or three volumes entitled *De pulchro et apto.*" The books, he adds, had disappeared, he knew not where. They have not been recovered, but from his recollections we learn that, probing the nature of beauty and its significance, he had come to the conclusion that it was of various kinds, and that, of these, one lay upon the surface of things and lived in line and colour—this was beauty *per se*, manifest to all; but that another kind existed, which differed altogether and called for deeper vision. This form appeared in structure as it lent itself to intention, as it embodied plan, as it revealed design.[1] Matter now became the instrument of the mind, the means to an end; it was plastic unto thought. And as thought took shape in the material, so beauty appeared as the apt, the adapted, the well contrived. Thus without gaining in weight, for shape cannot be weighed, the

[1] St. Augustine, *Confessions*, book iv, chap. xiii.

ponderable assumed potential, i.e. the potency which form confers. Our museums, anatomical and pathological, are filled with fragments of mortality; gruesome these may be to the eyes of the uninstructed, but they are incomparably beautiful to the eyes which see, for they declare the mind of the Great Designer. Reversing St. Augustine's words, might we not fittingly inscribe over the portals of these museums the words —*de apto et pulchro?*

To proceed, the heart and the vessels are parts of one whole, namely, the circulatory apparatus. To circulate its contents, the blood, is the purpose of this apparatus; and according as this purpose is achieved, so is the heart judged both in health and disease. During life and after death we must always have in view this test of structure, its functional adequacy, our aim being ever to visualize dynamically.

It is hoped that in the course of the argument certain views advanced will not be regarded as showing too much temerity, and that they will be considered on their merits or demerits alone, the injunction borne in mind: "Thou shalt not ask—who hath said this? but to that which is said give heed" (*Non quæras quis hoc dixerit, sed quid dicatur attende*). I allude here, in particular, to the suggestion that there is a definite order of sequence in the contraction of the ventricular system of fibres, this applying especially to the left ventricle; to the view that the early diastolic mitral bruit is due to a physical act, whereas the late diastolic mitral bruit is of purely physiological origin; to the description

of the aorta as a *transformer* interposed between the heart and the capillaries; to the mode of functioning of the aortic valve; to the means of estimating the relative power values of the auricles and ventricles (a subordinate chapter deals with this more at length); to certain points concerning venous pulsations and the interpretation of their tracings; and to other views which need not be specified further here.

It remains for me to express my gratitude in the first place to Professor Keith, F.R.S., Conservator of the Hunterian Museum, who, in addition to his kind reception of myself, showed me how to find my way amongst his treasures: he has my warm thanks. In the next place I must thank Dr. Helen Chambers, C.B.E., who, as Head of the Pathological Department of the Royal Free Hospital, gave me every possible facility and help in the prosecution of certain investigations which I began before the war; to Dr. Gertrude Gazdar also, at that time Assistant in the same Department, I wish to acknowledge much kind helpfulness.

Finally, I must record with many thanks my indebtedness to M. Gaston Doin (Librairie Octave Doin, Paris) for kind permission to reproduce Fig. 10 from Testut's *Anatomie Humaine*; likewise to Messrs. J. & A. Churchill, in respect of Figs. 15 and 16 from Peacock's *Valvular Disease of the Heart*, and Fig. 17 from Morris's *Anatomy*; likewise to Messrs. Macmillan & Co., in respect of Figs. 8 and 9 from Luciani's *Physiology*; and lastly to Messrs. Longmans, Green & Co., in respect of Fig. 1 from Quain's *Anatomy*.

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## CHAPTER IX

# THE HEART AS A POWER-CHAMBER

## CHAPTER I

### CONSIDERATIONS ANATOMICAL AND PHYSIOLOGICAL

MANY years ago two of our most searching intellects, Professors Tait and Steele, lent their powers to the study of "The Dynamics of a Particle." The act is calculated to invite to humility and admonish against presumption, yet here is one who would attempt the dynamics of a host of particles! A host, however, is not necessarily a crowd, rather does it suggest some form and degree of organization; it is in this sense that it is here regarded, that is, as an organic whole, a compound particle in fact, and it is only as a study of organized action therein that the problem is attacked. Even so, the attempt is made in the full consciousness of the difficulties which present themselves, and of the inadequacy of the investigator; moreover, it aims only at a consideration of the simpler issues. It must be added that it pretends to no completeness: it is a contribution and no more.

The cells which essentially characterize the heart, as an organ, are muscular fibres of a peculiar kind, which in certain respects seem intermediate between the fibres of voluntary muscle and the fibres of involuntary or plain muscular tissue. The peculiarity to which special attention should be directed is the way in which the fibre cells branch and interdigitate; the branching securing an interweaving or interlacement of the lines of force (contractile), the interdigitation a locking of fibre to fibre which must make for strength. The structure as it stands lends itself obviously to co-ordinate action.

In the case of the skeletal muscles the contraction of the fibres takes place between their two points of attachment, viz. the point of origin and the point of insertion, and the result of the contraction, if effectual, is to approximate these two points. It is otherwise with a considerable number of the cardiac fibres, the points of origin and insertion of which lie close together in the fibrous rings at the base of the heart, which surround the auriculo-ventricular orifices and the origins of the great arteries of the heart. The course and arrangement of these fibres, admittedly very complex and still imperfectly determined, may be described broadly as taking the form of looping curves, which, starting from the fibrous rings at the base of the heart, return eventually to these same rings. In this course they enclose the ventricular cavities. Now the point to draw attention to is that in contraction these fibres do not tend to approximate their points of origin and of insertion, as do the skeletal muscles, rather do they tend to separate them and thus keep

open and stretched the fibrous rings. Were it not so, they would exert a sphincter action which, in the case of the auriculo-ventricular orifices, would be superfluous, inasmuch as these are already provided with valves which close them, and by their closure determine the forward course of the blood; whilst in the case of the orifices of the aorta and pulmonary artery any supposed sphincter action would directly check the egress of blood and thus thwart the very purpose of the ventricular systole. Nature is not superfluous or conflicting in those other designs which have stood the test of time, and we may take it that, however complicated the disposition of the heart fibres, their combined action secures as one objective an open and taut fibrous ring at the base. But to be effective the fibres must shorten,[1] and since they cannot do so at the base, they must somewhere else in their course from and to the base. They do so: the looping curves shrink, the walls of the heart cavities become approximated, even to the obliteration of those cavities, as the ventricles empty themselves; but until this expulsion is completed, the intraventricular pressure caused by the systole keeps the heart fibres ever on the stretch, and so the pull on the fibrous ring is maintained. Relaxation follows abruptly; the semilunar

[1] The argument here developed has reference solely to those fibres which take their origin at the fibrous ring and come back to the ring for their insertion. This course does not hold for all the cardiac fibres and notably for certain bands which make up a considerable part of the thickness of the left ventricle and of the interventricular septum; these encircle the ventricular cavity, and by their grasp are perhaps the most powerful system of fibres in expelling the blood from the ventricle.

arterial valves recoil with a snap, and the relaxed fibres, protected from the effects of their own contraction, yield gradually to the gentle pressure exerted by the inflowing venous blood. The venous inflow is assisted towards the end of diastole by a contraction of the venous sinuses, i.e. the auricles. The auricular systole puts the last of the venous blood behind the auriculo-ventricular valves, therewith distending the ventricles and raising the intraventricular pressure sufficiently to float into position the auriculo-ventricular valves. The ventricles are now charged, ready for the systole which follows immediately.

To return to the fibrous rings at the base of the heart, these form part of what might be called a fibrous-tissue system, which system is as essential to the cardiac function as are the muscular fibres themselves; for muscular contraction is useless unless the muscle fibre be so attached that it can bring to bear the force of its contraction. The attachment may be such that one point is relatively fixed; in that case the other point yields and approximation takes place, the skeletal muscles exemplify this; such attachment is linear; or both points of attachment are equally fixed, in which case the disposition of the fibres must be more or less circular. This is exemplified on a large scale in the working of plain muscular fibre. In this case the condition for effective, i.e. work-producing, contraction is that a resistance lie within the circle of contractility. This is seen when the bowel passes on its contents by peristaltic action, and this same kind of action obtains when the

heart contracts upon its contents and ejects them.

Quoting from Quain's *Anatomy*,[1] we find the statement: "While in connexion with the large orifices at the base of the ventricles a mass of fibrous tissue and fibro-cartilage occurs, which in some animals, as the ox, is bony and is known as the os cordis. This central fibro-cartilage is placed in the angle between the aortic and the two auriculo-ventricular openings, and from it

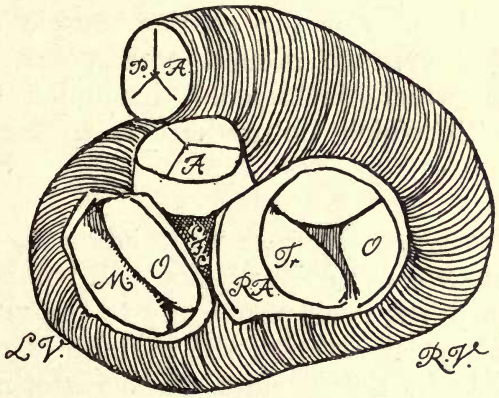


FIG. 1.—Base of heart, seen from above, the auricles removed. P.A. = pulmonary artery; A. = aorta; M.O. = mitral orifice; Tr.O. = tricuspid orifice; R.A. = a flap of right auricle; C.F.C. = central fibro-cartilage.

processes pass in various directions." These directions are enumerated as downwards "to meet the fleshy septum of the ventricles," and

[1] E. A. Schäfer and G. D. Thane, 1892, p. 367. The diagram is Dr. Allen Thomson's; it is found on p. 361. From it we see how central is the placing of the fibro-cartilage, and how from it and its extensions—the fibrous rings of the great orifices of the heart—the muscle fibres radiate in their downward and outward course. A flap of the right auricle covers up part of the central fibro-cartilage.

outwards " to form the bases of . . . the fibrous or tendinous rings of the auriculo-ventricular and arterial openings." From the rings the fibrous tissue extends into the segments of the valves, strengthening them. From this description it will be gathered what an important system the fibrous element constitutes in the structure of the heart. Here in this basal fibrous system we find the vantage ground from and towards which radiate and converge in great measure the lines of force which compose the ventricular systole. For the cardiac muscle and its task, this vantage ground corresponds to the πού στῶ, which Archimedes demanded for the lever with which he undertook to move the earth.

At this stage we may with advantage introduce a scheme of the circulation. It will be noted that it confines itself practically to the ventricles and their great arteries, the auricles being indicated only by dotted lines. The reason for this is that, physically considered, the ventricles are alone concerned in the circulation of the blood through the vessels. This question and the value or purpose of the auricles will be dealt with more fully later. In other respects the diagram explains itself. The shading of the walls has no bearing upon the course of the fibres, but in the interventricular septum a vertical line indicates the line of separation between the fibres of the two ventricles, respectively, and shows that practically the whole of the septum belongs to the left ventricle. The dividing line is drawn quite arbitrarily, i.e. diagrammatically.

The heart is a power-chamber; its projectile is its own contents; its field of projection the vascular system; its objective, physiologically considered, the capillaries of the tissues; but,

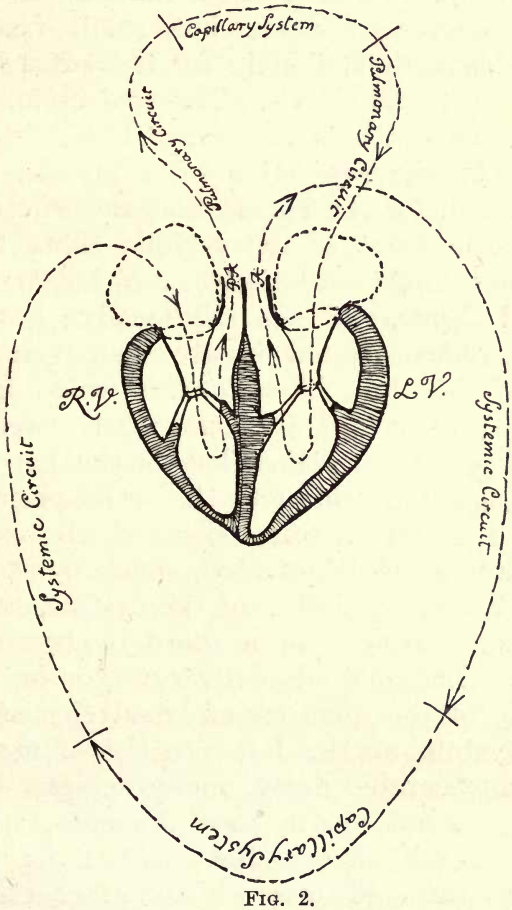


FIG. 2.

physically considered, its objective is its own cavities (auricular, ventricular), and until it has lodged its projectile there it has not found its mark. The projection of the blood from the

cavities of the heart is a recurring act more or less regular in time-sequence, in volume, and in force.

Relatively to the whole mass of blood in each circuit the volumes of blood in the ventricular cavities respectively are but small fractions. This holds most strikingly for the left ventricle and the systemic circuit. The projection, therefore, of the contents of each ventricle bears no likeness to the projection of a missile, e.g. a cannon ball, for the reason that the whole mass of blood in front of the systolic efflux forbids a movement of translation in any but the most minimal degree, and then only locally; a translation movement would involve a movement *en masse* of the whole blood, and it would demand enormous forces and absolutely rigid vessel walls. What happens then is this, that the elasticity of the arterial system, yielding to the act of propulsion, causes the rapid conversion of the systolic thrust into an elastic stretch, which same, by its recoil at the completion of the systole, compels the blood onwards in a more or less steady stream. The actual delivery at the right ventricle of the quantity of blood ejected by a given systole of the left ventricle must thus suffer considerable delay, many systoles taking precedence, but in due time it comes, and the delay does not alter in the smallest degree the fact that the sole force which has effected the delivery is that of the given ventricular systole: the term "projection," therefore, is wholly justified.

The right ventricle now takes up the completed left ventricular act, and in an exactly similar way transfers from the right to the left side of

the heart through the lesser trajectory of the pulmonary circuit. The blood is now back to its starting-point, having *obiter* passed through two sets of capillaries, the systemic and pulmonary, spending itself in the former, renewing itself in the latter. Between these two sets of capillaries, the function of the heart is to keep the blood in constant movement to and fro. This is the course of the circulation as traced by Harvey.

Comparing the two circuits, the systemic and the pulmonary, the former is vastly more extensive, as measured by length of vessel and multitude of ramification ; further, its arterial channels in general are narrower and thicker walled ; this applies specially to the arterioles, and we must note that these same are much more contractile, both passively, because they contain more elastic tissue, and actively, because the muscular element in their walls is more pronounced ; it must be added that, in correspondence with this last-mentioned fact, their vasomotor control is much more developed. The outcome of all this difference is that the resistance offered by the systemic circuit is vastly greater than that of the pulmonary. This resistance is of two kinds ; in the first place it consists of a passive, purely physical resistance such as persists after death—this may be regarded as a permanent, more or less constant quantity ; in the next place it consists of a potentially active resistance, which at any moment can be thrown into the circuit by arteriole contraction, *via* the vasomotor nerve fibres, and since this is essentially a vital manifestation, it must be termed

physiological as against physical. This is an occasional and an inconstant quantity, according to the degree of stimulation conveyed by the vasomotor nerves.

The sum of these two resistances, passive and active, in each circuit, gives the mechanical task of the corresponding ventricle, and hence the marked disproportion between the muscularity of the left and right ventricular walls.

Anyone who has watched the heart beating *in situ* will have been struck by the violence of the forces developed in the ventricular systole. But no act of propulsion can take place without an equivalent recoil, which must be met, whether by rigidity of fixture or by elastic play; and just as an *intrinsic point d'appui* has been shown to be essential to the effective contraction of the ventricular musculature, namely the central fibro-cartilage at the base of the heart, so it is certain that there is no less a necessity for an *extrinsic point d'appui* which shall meet and allow for the act of propulsion and its recoil, bringing back the heart at the end of each cardiac cycle to its starting-point, with the regularity and accuracy, almost, of the swing of the pendulum. This extrinsic *point d'appui* we find in the intrathoracic attachments of the heart, as also in a gentle pressure-support to which the heart as a whole is subjected. Let us look at these components.

Slung in the cavity of the thorax by the fibrous tissue of the mediastinum, by the parietal covering layers of the pleura proceeding upwards over the apices of the lungs, also by the great vessels which spring from the base of the heart and supply the

head, neck, and upper limbs (in particular the great arteries), the heart is stayed from above. Below, it is made fast by the attachment of the pericardium to the diaphragm, and well secured. On either side the roots of the lungs, with their pleural attachments and the radiating branchings of the pulmonary vessels, assist in the maintenance of the heart in its central position. To complete the maintenance, the air cushion of the lungs supplies a gentle yielding pressure, which practically surrounds the heart with its support (the study of a transverse section of the thorax with the organs *in situ* shows this at a glance) except below, and here the diaphragm, domed as it is by the external atmospheric pressure on the abdomen, as well as by the muscular tone of the abdominal walls, supplies the deficiency. Let us note of these attachments that whilst being firm they are amply elastic, therefore adapted to give the necessary play to the movements of the cardiac cycle [1]; and of the support, as it is supplied by lung and diaphragm, that in its gentleness it is adapted to leave unembarrassed those movements of small power which fill the cavities of the heart during diastole.

Can one conceive of any design more admirably contrived to secure at the same time freedom of action with continuous support?

From these general considerations we may now pass to a more special examination of the heart, and first, in order of precedence of action,

[1] It will be remembered here that the serous membrane which lines the pericardium and is reflected over the surface of the heart reduces to a minimum the friction of the heart on its surroundings as it contracts and expands.

the auricles present themselves. Considered physically, i.e. mechanically, the auricles are terminal venous sinuses which dilate passively to the venous inflow; considered physiologically they are power-chambers—that is the meaning of the muscular fibres in their walls—but in normal states their contractile powers are relatively small. Normally, during ventricular systole, and in quiet states of the body, as during sleep, there is probably no halt to the inflow of blood into the auricles; by "normally" is meant that the auriculo-ventricular valves are efficient in preventing reflux. But though there be no actual halt, there will be a gradual diminution in the rate of inflow as the auricle becomes distended and the intra-auricular pressure rises; should the tricuspid valve not quite hold, as is so often the case, apparently even in health, then the inflow into the auricle will necessarily become still more slow or may result in actual stasis, perhaps in slight retrogression. With the occurrence of ventricular diastole the point of lowest pressure is transferred to the ventricular cavities, and at once the auricles unload their contents. From this point onwards the inflowing blood from the veins will tend to fill both cavities, auricular and ventricular simultaneously, but ventricular distension will cease first because the walls are thicker and therefore less distensible: at this stage the auricle will go on filling whilst there is stasis in the ventricle. This brings us to that period of the diastole which immediately precedes the ventricular systole, when we find the ventricle imperfectly distended, yet the venous pressures incapable of effecting the requisite distension.

Now do we perceive the significance of the auricular muscular fibres; they contract; the blood is forced into the ventricle; distends it, and then the distension-recoil floats the auriculo-ventricular valve into position and puts it and the chordæ tendineæ lightly on the stretch. The ventricle is thus made ready for its own systole, the auricular systole having completed its task.

Before leaving this subject of the flow of the blood into the auricle and out of the auricle through the auriculo-ventricular valve, note should be taken that there are two points of time in the cardiac cycle, when the last-named flow, from auricle to ventricle, must show acceleration; the one is at the very commencement of the ventricular diastole, for then the auricle, which has been filling all through the ventricular systole, will be at its maximum of physical distension,[1] and the elastic recoil will consequently be at its maximum also—this recoil must take effect on the blood stream; the other point of time is just towards the termination of the ventricular diastole, for then the systole of the auricle will add its own impulse to the flow. This impulse is a physiological act which thus contrasts with the physical act of the elastic recoil occurring in the early diastole. The time-nomenclature of these accelerations is best kept in relation to the ventricular states of systole and diastole, and will be thus named post-systolic and pre-systolic, or early and late diastolic, respectively; either is equally descriptive. In passing let us note that in disease of the auriculo-

[1] Should any reflux from the ventricle have occurred, it will accentuate the auricular distension.

ventricular valves these times of acceleration are the times when murmurs are developed.[1]

Does the auricular systole serve any other purpose than that of filling the ventricle? It will be observed that its act is completed before the ventricular systole has begun, and whilst the aortic valves are still tightly closed, therefore no part of its drive can be contributory to the ventricular systole, but the whole of its force must be expended in distending the ventricle. May not, however, this act of distension, which is comparatively sudden, supply a stimulus which either starts the ventricular systole or helps to start it? We know that the sudden stretch of a muscle is an excitant to contraction —witness the knee-jerk—then why not here?

Wherever muscular tissue is present in the body, it is subject to the law of hypertrophy, according to which it tends to increase as the work which it performs is increased. The auricular muscle fibres exemplify this law in certain morbid states of the auriculo-ventricular valves which obstruct, more or less, the flow from the auricle or in any way add to the task of the auricular systole. From this point of view the muscle fibres of the auricles have a special value, which is of the nature of a reserve power to be called upon, *si opus sit.*

The fibrous rings which encircle the *a–v* orifices separate completely the muscle fibres of the

[1] It is true, mid-diastolic murmurs are described, but if they exist as a separate entity they have yet to justify their existence; for a murmur must mean an accelerated flow, and why the stream should suffer acceleration in the mid-diastole is, to say the least, hard to comprehend.

auricles from those of the ventricles; no contraction can therefore cross these rings [1]; the two hollow muscles are entirely dissociated. We may therefore ask here whether we have now come to an end of the auricular activities as an organ. Apparently it is so: on the one hand, acting as a venous sinus, it performs the humble part of passive conduction; on the other hand, by means of its muscular fibres, it acts as a force pump, and so completes the loading of the ventricle, preparing it for action. What else can it do as an organ, whose function is to pass on its contents; what else can one ask of fibro-elastic tissues and muscle fibres, for there are no other special tissues which belong to it *quâ* organ? Two functions, then, are present, passive conduction and active propulsion; have these equal value, are both essential? One is essential, that of passive conduction, for the blood must be returned to the heart; but as to auricular contraction it is otherwise, for whilst we may take it that it is essential to the *best* circulation of blood, it is certainly not essential to an effective circulation. This is but one instance—others abound—of the extraordinary balance of the heart's activities, which are so stabilized, so safeguarded that they are found able to carry on under conditions which excite one's wonder. And so we find that an auricle paralysed as to its muscle fibres may coexist with a practically efficient circulation, the subjects of the trouble being perhaps altogether unconscious of the auricular failure, and leading strenuous lives,

[1] See Quain's *Anatomy*, ed. Schäfer and Thane, vol. ii, pt. ii, p. 368; also Henle, *Gefässlehre*, 2nd ed., pp. 15–16.

including herein hard manual labour.[1] We have all seen such cases.

According to Henle it is generally accepted that the auricular capacities are somewhat less than the ventricular; but it does not profit to trouble about this nor as to whether the auricles empty themselves completely at the auricular systole, though one may say that it is very improbable that they do, seeing that the general opinion of physiologists holds that even the powerful ventricular cavities at the height of their contraction may fail to do so. All that it concerns us to recognize is that in each cardiac cycle the quantity of blood returned to the heart must, on the average, balance that which is expelled from it or there would be accumulation.

Physicists measure energy in terms of work done, e.g. foot-pounds, or whatever other units be adopted; can we estimate in this way the amount of energy contributed by the auricle to the sum total required for the circulation of the blood? If we were able to do this, and the like for the ventricle, we should be in a position to assign the exact relative value of auricle to ventricle in terms of numbers: we are speaking of these two chambers as power-chambers and nothing else. In default of such a method of valuing, we should arrive at an approximate relative estimate if we could count the muscular fibres in the walls of each chamber: this would rest upon the assumption that each muscular fibre was capable of the same effort and was called upon for the same effort; large assump-

[1] Cf. Sir James Mackenzie, *Diseases of Heart*, 3rd ed., pp. 227, 229, 231. Auricular fibrillation.

tions. Failing either method—and one need not expatiate upon the impracticability of each—a third method would be to weigh the muscle fibres of auricle and ventricle. This is the most feasible, though at best very imperfect, and I have endeavoured to approach the solution of the problem in this way. Reference to this will be made more at length after the ventricle has been dealt with, and at this stage it must suffice to say that the outcome shows what a relatively small part the auricle plays, in health, as a motive power. For all that, it is essential for the best circulation; and the moment disease steps in and puts an obstruction to the small powers, which are all that remain of the ventricular stroke by the time the blood reaches the great veins (which powers only just suffice to fill the auricle and ventricle under low tension), then it is of the utmost importance, for then it is that it comes to the rescue with its reserve of compensatory hypertrophy.

Comparing the two auricles, one with the other, in respect of their relative tasks, we come to the conclusion that the task of the left auricle should be the greater, because the greater thickness of the left ventricular walls should demand more force for their distension by the auricular systole; but when we come to look we do not discover any very noticeable difference in the thickness of the walls to bear this out. It would seem, then, as if the relaxed ventricles did not offer any appreciable difference in resistance to the amount of distension required. If so, the work done by each auricular systole would be approximately

equal, and in these circumstances it is probable that the septum auricularum, in so far as it contains muscular fibres, belongs equally to either auricle. The approximate equality of task of each auricle should lend itself the better to the synchronous action of the two systoles which actually obtains.

The disposition of the auricular muscular fibres is according to the following plan [1] :

1. Auricular fasciculi surrounding the mouths of the veins; these are continuous with the circular muscle fibres of the walls of the veins.

2. Fasciculi passing in loops from the *a–v* fibrous rings anteriorly, thence over the auricle to the *a–v* ring posteriorly.

3. Fasciculi common to both auricles and running parallel to the *a–v* groove; these bundles are most pronounced in the neighbourhood of the groove; they are more developed anteriorly than posteriorly.

The action of the first set will be sphincter-like, the tendency of which will be to prevent the blood wasting itself backwards into the veins; it will thus favour the forward movement required. The action of the second group will be to grasp and expel the blood into the ventricles. That of the third set will make for synchronous action of the two auricles; these fibres will of course co-operate with the loop fibres in expelling the blood. Is there an order of contraction? (cf. p. 128).

We now pass to the ventricles, and contrasting them with the auricles, we note that, whereas these

[1] See Piersol's *Human Anatomy*.

were little more than dilated venous terminals, the walls reinforced with a thin layer of muscular fibres, and the whole structure of the nature of a vestibule or antechamber, they, the ventricles, are the chambers to which the auricles were the approach, and wherein are generated the energies which circulate the blood. The origin and source of the circulation in each circuit are to be found here. Mechanically the ventricles are force pumps so designed as to maintain a flow of blood constant in direction. Muscular contraction supplies the motive power; valves ensure the continuity of direction.

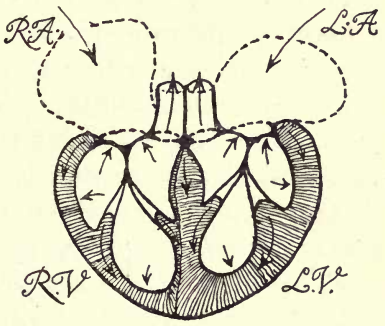

FIG. 3.

The schematic diagram, Fig. 3, is intended to show the heart in active systole: the *a–v* openings are closed by their valves, forced back into apposition by the compressed blood, which, finding no escape elsewhere from the grasp of the contracting ventricles, has forced open the semilunar valves of the aorta and pulmonary arteries. The arrows in the outer walls of the heart, and in the septum, show the direction of the pull of the muscular fibres. The arrows at right angles to the walls and to the segments of the valves show the direction of the resistance of the contained blood. It will be observed that the muscular pull starting from the base of the heart at the system of fibrous rings comes back again to the base, whether directly, or indirectly

through the septum ventriculorum, the musculi papillares, and the chordæ tendineæ. The contained ventricular blood everywhere under compression keeps the fibrous ring system at full stretch. This system has been purposely exaggerated at the periphery of the *a–v* openings, and where these openings abut on the origins of the great arteries. Comparing with Fig. 2, which presented open *a–v* valves, this diagram shows the heart as more globular in form, and the apex of either ventricle as much thickened by the act of contraction. It must be repeated that it is schematic only [1]: we shall have occasion to return to the question of the apical thickness of the ventricles in diastole and in systole.

The flow of blood into the auricle is continuous throughout the whole of the cardiac cycle, filling both auricle and ventricle during the ventricular diastole, but the auricle alone during ventricular systole; whilst continuous it is, however, very variable in rate of flow. In contrast the flow into the ventricle ceases absolutely during the whole of its systole. Perhaps the above statement is too absolute as to the auricle, for it may be that, even in health, there is a temporary arrest of the venous inflow during the brief period of auricular systole; and certainly in disease of the *a–v* valve, obstructive in nature, with consequent hypertrophy of the muscle wall of the auricle, we should look for temporary arrest during auricular systole,

[1] Another instance of the schematic representation is that in either chamber one musculus papillaris is made to spring from the septum, whereas in the left ventricle the septum does not give origin to any papillary muscle, and in the right it contributes very imperfectly.

and possibly more or less regurgitation into the venæ cavæ. When the *a–v* valve is unable to close its orifice effectively, whether through disease of the valve or dilatation of the heart, the auricle becomes directly subject to the ventricular systole, with more or less backward filling of the venæ cavæ or holding up of their venous current.

Every form of functional activity calls for a period of rest to make good the tissue disintegration and expenditure of energy which attend upon the activity. Accordingly the ventricular systole necessitates the diastole. But the manifold activities, now katabolic, now anabolic, which mark these states respectively, and which are present in every part of the body, never cease. When force is liberated there is tissue change, and when force is stored there is also tissue change. These changes are physical and chemical, formative and disintegrant, and since they are ceaseless, there must be a constant supply of formative elements and a no less constant removal of the products of disintegration; this demand is met by the circulation of the blood in a continuous stream. How is this continuity of flow secured?

The blood issues from the ventricle in a series of jets; it flows through the capillaries with a steady current; how is the conversion effected? By means of the elasticity of the arterial walls. From the origin of the aorta to the smallest ramifications of the arterioles a predominant structural element is elastic tissue, disposed in networks or in fenestrated membranes; this

elastic tissue, yielding to the propulsive force of the systole, transforms it into a stretch which suffices to extinguish wholly the intermittent movement of translation of the blood at the start. The even flow through the capillaries is a constant drain upon this stretch; it is met by the recurrent acts of projection of the ventricular systoles. The stretch of the arterial system as a whole suffices to fill the capillaries under tension, and likewise the veins, and its force is not wholly spent until we approach the heart and the negative pressure in the thorax. The elastic recoil of the arteries is responsible for the blood current through capillaries and veins, and we see here the significance of the predominant elastic element in the arteries.

Systole and diastole constitute the ventricular cycle. In the normal circulation, cycle follows cycle with a marvellous uniformity of force and of duration, both of the systoles and of the diastoles.

It has been said that the diastole makes good the systole; so it does, but it is probable that it is more than sufficient to do so, and that, as we so often see exemplified in biology, nature presents a large margin of reserve—in this case a large margin of time limit—in excess of the needful. This gives liberal scope for variation in the rate of sequence of the systoles without unduly embarrassing the heart. We observe this by noting that when the pulse rate is accelerated, the acceleration, if moderate, is chiefly at the expense of the diastole; the duration of the systole, on the other hand, is relatively unaffected. The systoles are thus placed nearer together.

When the pulse rate is greatly accelerated, the duration of the systoles is also encroached upon and the individual beat is more short-lived and more feeble.

In the foregoing, ventricular action has sometimes been spoken of in terms of both ventricles, at other times in terms of the left ventricle only; the matter is immaterial, for the ventricles work synchronously, and though we have not direct access to the pulse in the pulmonary artery and its branchings, as we have in the case of the arteries of the systemic circuit, yet we know

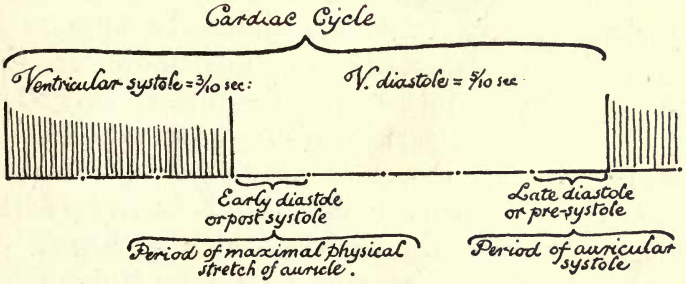


FIG. 4.

that the time relations of the right ventricle are identical with those of the left ventricle. These time relations are set forth in the accompanying diagram.

The whole cardiac cycle is here seen to be covered by eight-tenths of a second, three of these tenths being allotted to the ventricular systole, five to its diastole; all the cardiac events may be conveniently timed in relation to these two states of systole and diastole. In respect of the diastole, the diagram directs attention to two periods in particular, that which immediately follows upon the systole, and that

which immediately precedes it. Whichever nomenclature is adopted, systolic or diastolic, should be kept to for the sake of uniformity. The late diastolic period is stated to occupy about one-tenth of a second; it can be more exactly measured because it is marked by a definite act, that, namely, of muscular contraction. The early diastole cannot be thus delimited because it contains no definite act, but for convenience' sake the same period has been arbitrarily assigned here, viz. one-tenth of a second. The late diastolic period shows the blood moving at an increased velocity from the auricle into the ventricle, under the impulse of muscular contraction; it ends more or less abruptly at or just before the first sound. The early diastolic period also shows the blood moving at an increased rate from the auricle into the ventricle, under the influence of the elastic recoil of the auricle, which is then at its maximum of distension, i.e. stretch; this period has no precise demarcation; it tails off, the recoil subsiding gradually. In obstructive disease of the *a–v* valves, the late diastolic period becomes accentuated because of the auricular muscle hypertrophy necessitated by the valve obstruction, the blood moving with added velocity under the more powerfully contracting muscle; in the same disease the early diastolic period is liable to be accentuated, because there is very commonly a regurgitation from the ventricle associated with the obstruction, and this helps to distend the auricle to a more marked degree, thus accentuating the elastic recoil. Each of these periods tends then to show a murmur; sometimes one,

sometimes the other; sometimes there is a bruit in both periods. In a sense, as stated, the late diastolic bruit represents a physiological act, the early diastolic a physical act.

If we analyse the ventricular systole we observe that its onset is comparatively abrupt; all tracings show a steep ascent when the record is taken upon a slowly moving surface. Under similar conditions the relaxation of the muscle is sudden. This is well seen by the naked eye in the case of the perfused frog-heart beating in the oil chamber of the Roy-apparatus; we there observe the relatively rapid emptying of the ventricle, the muscle contracting and whitening with the expulsion of the blood; the maintenance of the contracted state for a brief but appreciable interval; then the sudden relaxation, the ventricle filling with stroke-like velocity. The apparatus in this latter respect does not copy the conditions of life, for whereas in life the distension of the relaxed heart takes place under a very low pressure (that of the venous inflow), in the Roy-apparatus the pressure of inflow is relatively high and the access of blood very free; all the more, however, does it accentuate the suddenness and completeness of the relaxation.

Clinically we get evidence of the same kind in the sharp click of the second sound which marks the recoil of the aortic blood upon the semilunar valves; the shortness of the sound giving evidence of the suddenness of the withdrawal of intraventricular pressure. In the relative length of the first sound, as compared with the second, we also get confirmatory evidence of the relative

length of systolic onset as compared with that of the diastole.

Recorded upon a more rapidly revolving drum the ventricular systole of the frog-heart inscribes itself as shown in Fig. 5. (The curve is taken from an actual tracing.)

Borrowing from the human heart the time relations of systole and diastole (it is only the curve of systole which has been copied from the frog-heart tracing), we have set out the cardiac cycle. Above the line AB the curve marks the development of energy (systole) and its complete

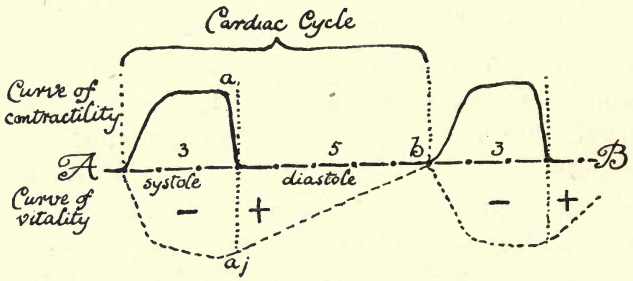


FIG. 5.

remission (diastole). Now during the whole period of the systole the muscular fibre is undergoing changes which are retrograde (katabolic); it is suffering disintegration and it is depleting itself of energy; on the other hand, during the whole of diastole the muscular fibre is engaged in changes which are restorative (anabolic), reinstating itself structurally and recharging itself with force. These changes in vitality are represented by the dotted curve below AB, and it will be noted that the curve of *devitalization* is the exact counterpart of the curve of *contractility*, and this is almost certainly true, since

molecular disintegration must keep exact pace with development of energy; but from the point $a_1$ onwards we know this much only, that re-integration is complete at the end of the diastole, as the dotted line shows. Whether the rate of repair is most active at the beginning of diastole, whether it keeps an even or variable pace, we do not know; it must suffice that when the point *b* is reached, restoration is complete. The signs, −, +, mark the preponderant katabolic and anabolic activities, respectively, which are present during systole and diastole.

The state of the cardiac muscle fibre during

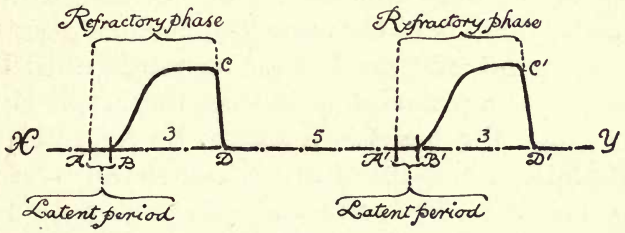


FIG. 6.

systole and diastole must be examined from yet another point of view, namely, that of its sensitiveness to stimuli applied at various points of time. In considering this we must start from the moment of application of an effective stimulus; this examination requires that the heart should have been rendered incapable of *spontaneous* beats, as by the ligature of Stannius.

In this diagram the horizontal line XY represents the line traced by the marker as the frog-heart, full but motionless, hangs in the oil chamber of the Roy-apparatus. In this state an electric stimulus (which experiment has found to be effective) is thrown in at the point of time A.

The response is not immediate, the heart waits until a point B has been reached; then the marker rises and traces the familiar curve BCD. From D onwards the horizontal line is again traced indefinitely (the heart being incapable of spontaneous beats, there are no time relations between systole and diastole) until an arbitrary point A′ is reached, when another stimulus is thrown in and, after a given pause, the marker begins to rise at B′, and the curve B′C′D′ is traced out.[1] It will be found that AB will be equal to A′B′. This period of pause after stimulation is called the latent period, because nothing obvious appears. Now let us throw in a fresh stimulus of the same quality and strength at any point of time from A onwards until C is reached, the probability is that there will be no response: the heart is found to be refractory to stimulation, and this state is called the "refractory phase." The refractory phase is of no fixed length; the term is specially and arbitrarily applied to the earlier stages of insensitiveness and generally is made to include the whole of the systole; but it tends to invade more or less the period of diastole until a sufficient interval has elapsed for the complete revitalization of the muscle fibre, then the same stimulus will repeat the identical curve B′C′D′, as shown. Should response occur between D and A′ (i.e. before complete restoration), the response will be less and less full according as the stimulus is nearer to D or C.

[1] In the diagram the second stimulus has been applied at a point when, in a spontaneously beating heart, a systole would be impending.

That these phenomena are due, in part, to a real insensitiveness, and not merely to an absence of contractile energy (which of course is diminished), is shown by increasing the strength of the stimulus ; for if we employ maximal stimuli instead of minimal we can curtail the period of refractoriness in proportion to the strength of incitation used.

These results are entirely in keeping with the findings set forth in the preceding figure (5), for we should expect that a tissue partially depleted of force and structurally lowered in vitality would declare this depreciated state by a lowered sensitiveness and a diminished response (should the stimulus elicit a contraction) until a sufficient time had elapsed to make good the damage done.

Reviewing these results, we perceive how the properties of the cardiac tissues would explain an irregularity of time rhythm, supposing that disease or a congenital structural flaw should disturb the normal distribution of stimuli at equal intervals ; would explain an irregularity in force rhythm, supposing that a like cause should disturb the normal distribution of stimuli of equal strength ; would prepare us for the occurrence of beats premature and immature, or the occurrence of actual suppression (intermittent action), supposing that from a like cause the stimulus should be of insufficient strength to become effective or, though adequate in strength, should happen to fall during the refractory phase.[1] The pulse gives us examples of all these vagaries, either separately or combined.

[1] A curious but very interesting observation was made when I was working with Dr. Ringer on the effect of drugs on heart

It has been said that the normal rhythm of the heart's action shows a marvellous uniformity (regularity) in its time- and force-sequence, each succeeding cardiac cycle being virtually the counterpart of its antecedent; so much is this the case that when we meet with any serious divergence from this orderliness we are apt to infer a corresponding failure of the circulation itself. But this inference is not justified by experience, for we meet with cases from time to time which, whilst showing an extreme disorder of the cardiac rhythm, do actually present a circulation which is adequate for the practical purposes of an active and useful life prolonged to the threescore and ten years limit, or beyond. And when we look at the matter theoretically, we are driven to conclude that, where a sequence of acts is so frequent as that of the heart-beats, an average will be struck in which the delayed beat is made up for by the premature beat, the small and weak impulse by the more vigorous act, and that this average will manifest itself in a blood pressure which is practically uniform in its level. The disorder of the individual beats

response; it was this: a stimulus adequate to produce a full contraction was thrown into the Stannius'd frog-heart, and then, *whilst still within the latent period*, a second adequate stimulus. In many cases the second stimulus produced no visible effect whatever, the normal curve appearing at the end of a latent period of normal length; but in certain cases the normal curve was wholly suppressed. This result came out too often to be an accident, and the only suggestion I could propose was that two stimuli falling upon a sensitive tissue too near together may interfere the one with the other, and cancel each other, in the same way that two identical undulations meeting in opposite phases may cancel each other, as may happen, e.g., in the case of two waves of light.—H. S.

thus loses itself in the uniformity of the elastic arterial stretch, which, being compounded of a multitude of beats, presents their sum total but not their differences—it is not sensitive enough to do that, the momentum involved being too great.

Let us now pass to a consideration of the individual features of the two ventricles. Hitherto we have treated them somewhat indifferently as one or the other, or as one representing the other; now we must give them separate attention, for the first thing which strikes us when we dissect the heart is the disparity between the two ventricles; disparity of form, the somewhat crescentic cross-section of the right ventricle as against the circular cross-section of the left ventricle; disparity of thickness of the walls of the chambers, thin on the right side, thick on the left—we note here that the septum associates itself, in this respect, with the left ventricle. Then when we pass to the circuits served by each we note the great disparity which exists between them, the small resistance offered by the pulmonary circuit as compared with that which the systemic circuit presents. Everything, in fact, suggests duality, and that we have before us two separate ventricles, not one ventricle two-chambered: two hearts, not one heart. This is so; and yet so intimate is the association that the two move as one piece.

Then how is this unity of action obtained?

It is obtained by an exact proportioning of the relative loads (blood-mass and circuit-resistance) to the relative contractilities, so that in the unit of time the same quantity of blood is delivered

across the longer and shorter routes : this involves, of course, unequal blood pressures and unequal velocities, but both accurately correlated.

It is further obtained by that economy of material and of power which Nature exhibits so remarkably ; for why have complete separation when juxtaposition will permit one party wall to serve two chambers, delimiting each, though essentially belonging to one only of the two chambers. How this astonishing feat is performed we shall see, and this brings us to the structural features of the two ventricles.

In their main outlines the mechanism and working of the ventricles have already been sketched, as power-chambers ; the power residing in the muscular fibres which compose the walls of the chambers, which power finds its *point d'appui* in the fibrous system centring in the base of the heart, and to which belong the fibrous rings surrounding the auriculo-ventricular openings and the orifices of the great arteries, the aorta and pulmonary artery. The arrangement of these fibres must now be discussed.

If we look at the older dissections of the heart we find ourselves up against a great complexity, which is manifest primarily in the variation of the course of the fibres as we uncover these at different levels in the thickness of the walls (see the diagrams in Quain's *Anatomy*). Now as to the unravelling of this complexity we must not ask too much, and for this reason, which meets us at the outset, that anatomists are agreed that the individual bundles of fibres rarely, if ever, remain single, but on the contrary are by their branchings in constant intercom-

munication and interlacement; and that the like holds true for the layers or sheets of fasciculi. In consequence, whilst a general parallelism of the layers may be an observable feature, it is found to be a shifting parallelism as the heart walls are penetrated.[1] The only interest to the physiologist which the arrangement of the heart fibres offers is in respect of the light which it throws upon the powers developed by the heart muscle; what he wants to trace is the lines of force, and it must be at once apparent how insolubly difficult must be the pursuit of these lines through the intricate interweavings and subdividings of fibres and fasciculi and layers, which the heart presents, if our search is with any expectation of accuracy of detail. But one conclusion comes out prominently from amidst the tangle, namely, that this very complexity and intimacy of interweaving must add enormously to the strength of the structure and its solidarity of action.

Accepting these limitations of investigation, it may be said that certain general structural features do appear through the complexity; they may be set down as follows:

1. The presence of one system of fibres, of front-rank importance, which take origin at the base of the heart from the fibrous structures already described, and proceed in looping curves around the ventricular cavity to find their way back ultimately to the fibrous tissue system of the base. This insertion into the base may be

[1] Consult in particular "Muscular Architecture of the Human Heart," Franklin P. Mall, *Amer. Journ. of Anat.*, vol. xi, 1910–1911; also *Quain's Anatomy*, Schäfer and Thane.

direct or indirect, in the latter case *via* the musculi papillares and their tendinous prolongations (the chordæ tendineæ) to the edges of the valves, and so to the auriculo-ventricular ring.

The course of these fibres is more or less oblique, some of them running almost vertically from the base towards the apex of the heart—this lie is best seen over the left ventricle; others near the base run with but small divergence from the line of the *a–v* sulcus. Many of these latter pass


FIG. 7.—Apical vortex or whorl of left ventricle seen from below. (Borelli, *De Motu Animalium*, 1681; *Tabula decimasexta*, Fig. 4. For right and left vortices see Fig. 7A.

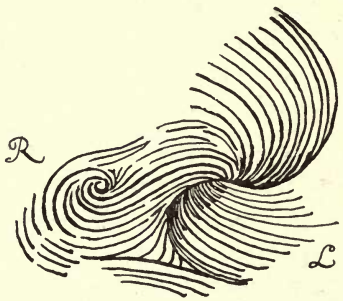


FIG. 7A.—Right and left vortices of apex of heart from Franklin P. Mall's "Muscular Architecture of the Human Heart," *American Journal of Anatomy*, vol. ii, No. 3, March 1911. By kind permission of the publishers, the Wistar Institute.

from the right ventricle to the left across the septum, and therefore are *common* to the two ventricles. Other of the system of fibres, and a considerable proportion of those whose greater obliquity of course brings them near the apex, dip in there in a double vortex or whorl and, penetrating the heart wall, proceed along its inner surface directly upwards to the base or indirectly upwards in the musculi papillares to the edges of the valves. The main vortex is at

the left apex; the vortex at the right apex is quite subsidiary.

The fibres entering into the whorl or many of them seem to take a turn upon themselves in the act of being reflected on to the inner surface of the heart, forming figure-of-eight curves,[1] whilst others keep to the form of the simple V-shaped loop.

Let us at this stage notice how the apex of the heart is fortified by this arrangement of the whorl, which confers the great mechanical advantage of the torsion or twist, for we shall have occasion to note that *per se* it is a weak spot.

It is of great interest to learn that Borelli, the contemporary and colleague of John Finch at the University of Pisa, during the latter half of the seventeenth century, showed an intimate knowledge of the structure of the heart, in particular of the course of its muscular fibres, as well as a keen appreciation of the complex problem of its working. His figures of the lie of the superficial fibres would serve present-day purposes; he recognized the apical whorl, and points to the penetration of the fibres at this spot, variously bent and interwoven, and their subsequent distribution over the inner walls of the ventricles—certain of the fibres returning to the base of the heart, certain others terminating in and constituting the musculi papillares, which give attachment to the funiculi of the bicuspid and mitral valves. He likens the whole heart to a ball (glomus) of wool or yarn, hollowed out

[1] See Bonamy et Beau, *Atlas d'anatomie descriptive*, Tome II, Cœur.

within ; and though his views as to the expulsion of the contents of the heart, not so much by the shortening of the fibres as by their thickening (swelling) in the act of contraction, would hardly find present-day acceptance, yet the general purview which he presents is most striking and illuminating.[1] His words are worth quoting: "It befell me first to observe this marvellous structure, whilst at Pisa in the year 1657 ; the illustrious Malpighi being present. Subsequently I learned that others had made the same observations. At length the renowned Lower, also Laurentius Bellinus, traced out the exact structure of the heart by resolving the perplexing interlacement of the fibres into the likeness of a clew of yarn." [2]

A feature in this system of fibres is their curving, oblique, or even spiral course, as we follow them from the outer surface through the apical whorl to the inner surface of the heart. Another feature is their relation to the fibrous ring system at the base of the heart, from which they arise, and to which they return either mediately or immediately. They might, therefore, be named appropriately the spiral or basal system of fibres.

We pass now to system No. 2, which shows us an order of fibres contrasting markedly with system No. 1 in that their course is circular. These fibres are situate between the external layers of the spiral system and the internal, reflected layers of the same system. They are restricted chiefly, if not wholly, to the left

[1] Giovanni Alfonzo Borelli, *De Motu Animalium, pars altera ; opus posthumum,* A.D. 1681. Cf. pp. 88–90, 91–92, 103–109.

[2] Borelli, op. cit., p. 90 : "*Hanc mirabilem structuram,*" etc.

ventricle. In Bonamy and Beau's atlas they are described as the fibres proper of the left ventricle, occupying the intermediate position just mentioned, and there forming a series of circles, one within the other, around the cavity of the ventricle. This system, apparently, has no connexion with the fibrous tissue at the base of the heart. Krehl also insists upon this independence of the circular fibres; the circles completing themselves and being wholly unconnected with the tendinous structures of the base: thus they encircle the ventricle as the muscular fibres of the middle coat of the arteries surround the lumen of the vessel. This system is "Krehl's Triebwerk," his "driving mechanism." Borelli seems to have recognized the transverse direction of the left ventricular intermediate fibres as a fact,[1] though I do not discover that he separates the system from that of the spiral system in the same definite way.

In this it is possible that he was both wise and prescient, for though the more generally accepted view of the independence of the circular system of fibres has prevailed in the past, and has been reasserted in the present by Krehl,[2] there is a school of anatomists, including Ludwig and Gerdy, and to which Mall adheres, which maintains that these fibres are in fact attached to the fibrous structures of the base of the heart, and

[1] Borelli, op. cit., p. 105: "*Praeterea paries intermedius cordis, constans ut plurimum ex fibris transversalibus. . . .*"

[2] It is a pity that Krehl has given to these circular fibres the special name of "Triebwerk," for, after all, their contraction does but co-operate with the contraction of all the other fibres of the spiral system, which consequently have an equal claim to belong to the driving mechanism of the ventricle as a whole.

that they are not truly circular, but spiral. The arguments in support of this are well given in Mall's paper,[1] in which we find figured a band of fibres termed by him the deep bulbo-spiral band, which would correspond with Krehl's Triebwerk. We there see that the spiral course departs so little from the horizontal as to be circular to all intents and purposes in its *constrictive action*, and the difference between the two schools resolves itself rather into that of the dependence or independence of the system in respect of the fibrous structures of the base. This question has its developmental interest, but we must leave it there for the anatomists to decide, and, since it is the mode of working which essentially concerns the physiologist, must point out that either view gives us a powerful band of fibres whose working is practically circular and sphincter-like—the name, therefore, of the circular system of fibres should be retained at any rate for the present.

The extent of the circular band varies much in different hearts, according to Mall. In the new-born and in young children it is very insignificant; which indicates that during growth of the body generally it must increase more rapidly than the other heart muscle bundles. This is of interest if we regard the rapidly increasing demands upon the left ventricle (during the early stages of growth of the child) to meet the enormous increase of resistance which the corresponding growth of the systemic circuit must involve. It also accentuates the importance of the part played by this system of fibres in the ventricular systole.

[1] Op. cit., p. 240.

The depth of the band varies in different individuals, and it falls far short of the apex.

The above description of the course of the fibres of the heart is taken essentially from the left ventricle, but the right ventricle shows an arrangement quite similar in respect of the system No. 1, though upon a much more slender scale, especially as to the apical vortex. In respect of system No. 2 opinions vary; some maintaining that the intermediate circular fibres are represented, though excessively thinned out [1]; others that they are not present at all: in any case they will not count for much functionally.

Leaving now the arrangement of the muscular fibres of the heart, let us consider their mode of action and put the question: Do they come into action at one and the same moment in the ventricular systole—we are referring to the two systems of fibres—or do they act separately and in a fixed order? The answer is, that there is reason to believe that the two systems act in a definite sequence, and that the longitudinal looped and spiral fibres precede the circular fibres in their contraction. The grounds for this view are as follows:

There are two stages in the act of systole which must be kept distinct. The first stage must be regarded as preparatory; it shows us a gathering intracardiac pressure under the increasing grasp of the muscular fibres: during this stage the physicists will tell us that no work is done, the whole of the muscular force spending

[1] Bonamy et Beau, loc. cit.: "Les fibres propres, intermédiaires aux fibres réfléchies, sont excessivement minces; elles décrivent des cercles plus ou moins complets."

itself in the development of a tension which is steadily rising. This stage is occupied then by the storage of potential force, as tension ; tension of the whole fibrous-ring system of the base, as seen in the taut auriculo-ventricular orifices and orifices of the great arteries ; and tension within the heart as seen in the stretched *a–v* valve segments straining upon their attachments, the chordæ tendineæ, and communicating their pull to the musculi papillares, which in their turn react. The obstruction determining all this accumulation of force is the resistance opposed by the blood pressures within the great arteries acting upon the semilunar valves.

The second stage is marked by the forcing of the semilunar valves and the ejection of the ventricular contents into the great arteries. In this act the potential which had gathered becomes kinetic as momentum and work is done by the heart muscle.

Now as to these two stages we know for certain that the system of spiral fibres comes into action with the first beginnings of the first stage, because from that very moment the intracardiac pressure begins to rise and therewith, immediately, the *a–v* valves are pressed upon, so as to force them back towards the auricles. Such movement, however, if it took effect, would *pro tanto* frustrate the blood pressure rise, and to avert this the musculi papillares pass into instant contraction, resisting the pull. It may be set down, then, as axiomatic that to make possible the rise of intraventricular pressure, the auriculo-ventricular valves must be held from the very beginning of systole, and must be held increasingly as the pressure tends

upwards. But we have seen that the musculi papillares are an integral part of the system of spiral fibres, specially designed to act in concert with and make effective the grasp of the rest of the system upon the contents of the ventricle.

It may be objected that the intracardiac blood pressure does rise in spite of a regurgitation through the *a–v* valves, when disease has rendered these incompetent. Yes it does when the regurgitation is not excessive, and even then it is thanks to the effective operation of the musculi papillares in holding as much of the valve as remains intact, but it must be added that, where the regurgitation is appreciable, the failure of the intraventricular pressure to rise adequately is exactly proportional to the extent of the regurgitation.

Admitting, then, that the spiral system of fibres enters into contraction at the outset of systole, and, indeed, thus constitutes the systole in its first stage, what about the circular fibres ? Do they also contract at the same moment ? I think the evidence is as clear that they do not. In proof of this let us look at Fig. 2 ; it will be seen there that the apices of both ventricles are attenuated relatively to the thickness of the rest of the cardiac walls and notably of the walls towards the base. This was first borne in upon me when endeavouring, in the course of certain weighing experiments, to peel off some of the subserous fat on the apices ; to my surprise the thinnest shaving laid open the cavity of the right ventricle. Passing to the left ventricle, I narrowly escaped doing the same thing. This happened to be a

morbid specimen, but the examination of healthy hearts showed that the finding was no morbid phenomenon, and that there was habitually a surprising disparity between the thickness of the walls at apex and base. This apical tenuity had not escaped the observation of the anatomists; thus Henle makes special mention of it, and says that the thickness of the wall of the left ventricle may hardly attain to 1 mm. at the apex.[1] If we compare this with a thickness of 1·5–1·75 cm., i.e. fifteen to seventeen times as great, a record not out of the common for measurements towards the base, we shall have to admit that a simultaneous contraction of every part of the walls of the ventricle is hardly credible. In Quain's *Anatomy* we find like reference to the disparity of thickness between apex and base; it is there stated that the greatest thickness of the walls of the left ventricle is distant from the base about one-fourth of the length of the heart: Quain adds that the apex is the weakest part of the ventricle.[2] Borelli draws attention to the same thing,[3] taking us thus back to the latter half of the seventeenth century.

These thin spots, it is true, are often only tiny pockets in amongst the trabeculæ and columnæ carneæ, which are so marked a feature at the apices; but at the commencement of systole the pockets would be still in communication with the general cavity of the ventricle, and therefore would be subjected to the full force of the contraction of the more powerful parts of the heart:

[1] Henle, *Gefässlehre*, 2nd ed., p. 45.

[2] Quain, op. cit., p. 362.

[3] Loc. cit., p. 103: "*Verticis verò paries valdè subtilis est.*"

how could these thin spots bear the strain? Nay, more, during this stage, the thinner parts pitted against the thicker parts would be actively thwarted in their endeavour to shrink and close up their loculi in preparation for the further strain.

The conclusion, then, is that these *loci minoris resistentiæ* form the most cogent argument against the simultaneous contraction of the whole muscular apparatus of the ventricle at the outset of the systole, and for a preliminary stage of contraction of the spiral system of fibres, which would prepare the apex to meet its task. How this would be effected we can readily perceive if we recall the structure of the apical vortex and visualize the effect of the shortening and swelling of the muscular fibres composing the intersecting system of columnæ carneæ there present; every pocket would be squeezed empty by the screw-like twist impressed upon the apex, which tightened, retracted, and solidified would present a resistance equal to any demands made upon it.[1] (Figs. 7 and 7A.) It is assumed that this state would be complete towards the end of the first stage of the systole, and that then the circular fibre system would come into co-operative action with the spiral system and force the semilunar valves. This, the second stage, would last until the great arteries had received their apportioned load, both systems of fibres working together until the end of systole.[2]

[1] Those who have watched the pulsating frog-heart will have observed how the ventricle, which in some two-thirds of its length, from the apex upwards, is little else than a spongework, shrinks, whitens, and becomes very compact during the systole.

[2] It is of great interest to learn that electric examination of the contracting ventricles discovers two points at which con-

This argument will hold with equal force whether the circular system of fibres be a special system having no connexion with the fibrous base, as the older writers have maintained; or be only a special part of the spiral system, and having therefore its origin and insertion in the basal fibrous structures; for by virtue of its circular disposition it would still be a special system in its mode of working, nor is there any reason why the distribution of nerve stimuli should not follow a definite order of sequence throughout the layers of fibres of one and the same system.

What has been said above applies *in toto* to the left ventricle; but since the right ventricle is either devoid of the circular fibres altogether, or contains them only in such small proportion that their action is practically negligible, we must regard its basal system of spiral fibres as adequate, without reinforcement, to deal with the smaller resistances opposed by the lesser circulation. The main features of the distribution of the fibres of the spiral system of the right ventricle appear to be the same, to all intents and purposes, as in the case of the left ventricle, but on a much smaller scale.

traction appears to take precedence, viz. one point near the apex to the right of the septal furrow and another at the extreme point of the whole heart, i.e. over the left apex—see Starling, *Human Physiology*, 3rd ed., p. 996. In a small-type paragraph on the same page, it is argued that the contraction of all parts of the ventricle must be simultaneous, otherwise, should the base contract before the apex, the uncontracted part must yield. This is of course true, but it does not meet the full requirements of the case, since it takes no account of the disparity of apex and base as to thickness. With simultaneous action the thin apex must yield to the thick base.

The next question which arises concerns the septa of the heart.

The interauricular septum calls for first consideration and may be soon dismissed, for the forces involved in the auricular systole are quite small, and their value on either side of the septum nearly equal in health. The septum contains muscular fibres (they are well figured in Henle's *Anatomy*), and in the auricular systole they will participate, but it is probable that they play but a small part in a comparatively unimportant act and that, such as it is, their part belongs equally to either auricle. The septum serves the office of a party wall in dividing the impure from the purified blood, and this will about sum up its action.

It is quite otherwise in the case of the interventricular septum, for here the forces involved are very considerable, and at the same time the disparity between them, as they are developed on either side of the septum, is extreme. It is obvious that as in the case of the auricles, so here, the septum will act as a party wall in keeping separate the venous and arterial bloods; but it is no less obvious that in its participation in the act of systole, either it must range itself exclusively on the one side or must share its action in due proportion with each ventricle.

What are the anatomical facts?

1. That in its thickness the septum associates itself with the left ventricle.

2. That in its direction, its curvature, it likewise associates itself with the left ventricle, completing the oval or circle which the outer walls describe.

The accompanying Figs. 8 and 9, from Luciani's *Physiology*, vol. i, pp. 183, 185, are after Krehl; they show well these two features. In Fig. 8 the cross-section of the diastolic heart shows the left ventricle nearly circular, as completed by the septum; the right ventricle being roughly D-shaped.

In Fig. 9 the cross-sections of the systolic heart at different levels show both cavities nearly emptied of blood, but the oval or circle of the left ventricle is well indicated without a break at the septum—also the crescentic cross-section of the right ventricle.

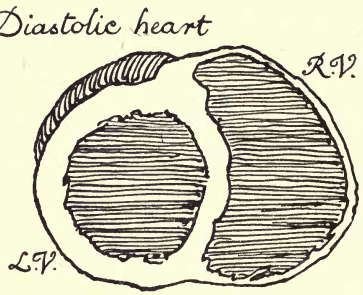


FIG. 8.—Section at junction of upper and middle thirds. The cross-sections of the trabeculæ and columnæ carneæ of the right and left ventricles are omitted.

The diagram Fig. 10, from Testut,[1] shows, perhaps, most convincingly the solidarity of the septum with the left ventricle. The cross-sections of the musculi papillares are prominent.

The crescentic form of the cavity of the right ventricle is also very apparent.

It has been stated that the existence of the circular system of fibres in the walls of the right ventricle is in doubt; some authors stating that they are present, but in excessively thin layers, others denying their presence. Admitting their existence, they must be confined to the outer walls of the ventricle, for they could not be

[1] *Anatomie humaine.*

present in the septum, since any fibres so disposed in the septum, i.e. transversely to the axis of the ventricle, would be convex towards the right ventricle, concave towards the left, and their contraction would work constrictively not upon the contents of the right ventricle, but of the left. We may therefore add to the anatomical facts 1 and 2, a third, in proof that functionally the septum belongs to the left ventricle, viz. :

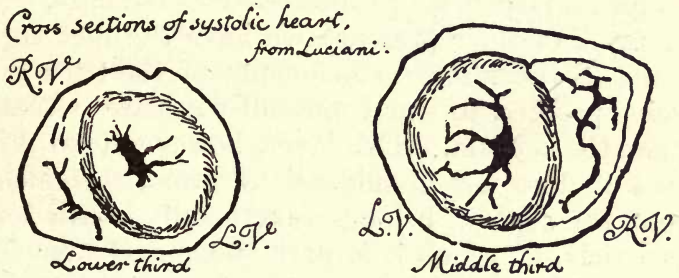


FIG. 9.

3. That the curve of the septum, convex towards the right ventricle, forbids the presence

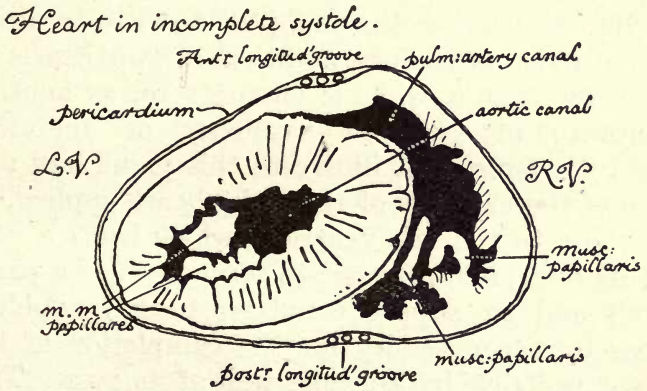


FIG. 10.

in it of any circularly disposed muscle fibres capable of working for this ventricle.

It must now be added, in apparent contradiction to our argument, that the septum does contain a thin layer of superficial fibres belonging to the right ventricle, but these fibres appertain to the spiral system, and are reflected from without inwards at the apex and possibly also along the interventricular sulci, anterior and posterior, where fibres from the outer surface have been found to dip in. These reflected fibres then run a longitudinal course upwards towards the base, and in proof of their presence we have the fact that some of the minor attachments of the tricuspid valve proceed to small musculi papillares situate upon the septum. This layer, however, is so thin that it does not invalidate the broad statement that the septum belongs functionally to the left ventricle, of which it is part and parcel; whilst to the right ventricle it serves only as a party wall, though, in addition, it may lend its mechanical support to certain of the spiral fibres reflected obliquely upwards and distributed internally.

Before leaving this subject I wish to draw attention to this incorporation of two hearts in one, for that is what it amounts to, as another instance of Nature's economy of material. Testut's figure will illustrate this point best: it shows the crescent of the right heart applied, as it were, to the left ventricle, which lends a part of its own circumference to serve both as a party wall and to supply counter-pressure, avoiding thereby the necessity for the completion of the right ventricle by a proper wall of its own. This part of the left ventricle contributes no part to the expulsive action of the right ventricle. The appression of the right ventricle takes place by

carrying an outer layer of fibres (of the spiral system) round the right ventricle and on to the left ventricle, where the fibres find attachment superficial or deep.

It will thus become manifest that the grasp of this common layer will serve not only to prepare both ventricles for the final act of expulsion in the manner already explained, but continuing its grasp it will contribute to the effort of the circular system of fibres of the left ventricle, which now comes into play, and it will suffice, on its own, to empty the right ventricle. It is to be noted that whereas the expulsive act of the left ventricle is largely due to a concentric contraction of the circular fibres, that of the right ventricle is effected wholly by appression against the interventricular septum (see diagram).

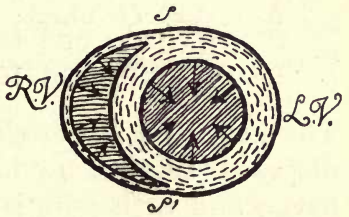


FIG. 11.—Scheme showing modes of contraction of the right and left ventricles—S, S′ mark the septal grooves.

## CHAPTER II

### THE RELATIVE POWER-VALUES OF THE AURICLES AND VENTRICLES; THE WORKING OF THE CARDIAC VALVES, IN PARTICULAR THE PROBLEM OF THE AORTIC VALVE

THE heart is a power-chamber. A longitudinally disposed septum divides its cavity into two parts, and each part is further divided into two by transversely placed valves. There result four compartments—two auricles, two ventricles—in each of which power is developed separately. The force-developing tissue is that of the muscle fibres laid down in the walls of the compartments. The act of the systole of the whole heart is to circulate the blood through the two circuits, the greater and the lesser, a purely mechanical act—how do these cavities individually contribute to the combined act, and how shall the quotum of each be determined? The first point to note is that the office of each compartment is exactly the same qualitatively, viz. the development of force: there is nothing else to develop. Accordingly the determination required is the exact amount which belongs to each—it is a mere question of how much. These cavities, then, will take rank in the order of their numerical values.

Regarded thus, are we prepared to allow to the auricles the somewhat predominant consideration which they have received of late years?

The answer must be, certainly not, i.e. as mechanical factors in the circulatory composition of forces.

We have already touched upon this question when dealing more generally with the auricles, and a brief recapitulation of certain main points may perhaps suffice at this stage; let us then note:

1. That the auricular muscle fibres cease abruptly at the auriculo-ventricular fibrous rings. The effort of each auricle is therefore confined strictly to itself.

2. That this effort takes place, and is completed, before the ventricular systole begins, *a fortiori*, before the semilunar valves are forced. Therefore nothing of the auricular effort crosses the semilunar valves to contribute to the circulation in the greater and lesser circuits.

3. That the whole force of the auricular systole is spent in completing the ventricular load by a maximal distension of the ventricle. Incidentally it has been suggested that the mechanical stimulus of the stretch of the ventricular walls might serve to initiate their systole.

4. That the pressures with which the right auricle has to deal will be very low seeing that, during the greater part of the ventricular diastole, both auricle and ventricle are filling under venous pressure alone (this pressure is put at 3 mm. Hg for the inferior cava and 0 to –8 mm. Hg for the large veins at the root of the neck[1]); the chief effort, therefore, required of the auricular muscle, towards the end of the diastole, will be that needed to distend the *thin, relaxed* walls of the right ventricle: this force must be very small.

[1] Cf. Starling, op. cit., p. 922.

The like will hold in the case of the left auricle, but the much thicker walls of the ventricle will call for a somewhat greater presystolic effort. The difference is probably only slight normally ; in disease a great disparity may exist.

5. That experiments upon the isolated heart, e.g. the excised heart of the frog, have shown that a vigorous ventricular action can be long maintained independently of any auricular co-operation ; and that, in man, observations in disease have taught the same thing, namely, that an adequate circulation may continue indefinitely though the auricle be completely paralysed.

The conclusion is that in the order of power the auricles will take quite low rank, and that, though necessary to the best circulation, an adequate second best may obtain without their help. In certain forms of disease, however, the auricular muscle may become very important, in particular in mitral valve obstruction, where the proper filling of the left ventricle is impeded. Compensatory hypertrophy then comes to the rescue.

We now come to the ventricles and their dynamic values. We have seen that at need the low pressures in the great veins near the heart will of themselves suffice to fill the auricles and ventricles in effect. These low pressures are all that remain over of the ventricular stroke, which, starting with an intracardiac left ventricular pressure of 130–150 mm. Hg (in the dog), generates

an average pressure in the aorta of 100 mm., whence it falls gradually, until in the capillaries it reaches 15–40 mm., and in the emerging small veins, declining still further, may subside to close upon zero, as the current nears the right auricle. That such low pressure can still fill the right side of the heart is proof that negative pressures obtain within both auricle and ventricle.[1] These negative pressures are due to the elastic distension of the lungs and have nothing whatever to do with the relaxation of the ventricle, for it is an immense relief to find that at last physiology has come into its own, and has got rid of negative intraventricular pressure *as a definite ventricular act.* The view, indeed, that a relaxing ventricle possessed resilience and could exert suction seemed so improbable on the face of things, so out of keeping with the simplicities of nature, that one could only accept the teaching under mental protest, feeling convinced that the phenomenon, as recorded, was much more likely to be due to instrumental shortcomings, which all instruments obeying Newton's law of inertia must tend to develop. We now learn that a more perfect apparatus, which diminishes enormously the amplitude of vibration of the membrane of the tambour, and gets rid of the inertia of the customary inscribing lever, by using the beam of light instead, gives a curve which may descend to, but never falls below, the zero line: such is the manometer of Piper.[2]

In health the ventricular systole alone suffices

[1] The argument here is applied to the systemic circuit. It holds certainly during the inspiratory phase of respiration.

[2] Starling's *Physiology*, 3rd ed., p. 941.

to bring the blood through the vessels of its circuit back to its starting-point in auricle and ventricle; but it is advantageous, in view of the very low motive power actuating the blood current towards the completion of the drive, to reinforce the act so as to meet impediments which may arise even in health during the rough and tumble of circumstances. To this end muscular fibres are laid down in the auricular walls, which, coming into action just before the systole of the ventricle, complete the filling of that chamber, float into fuller apposition the segments of the *a–v* valve, and confer on muscle fibres and tendinous structures that slight stretch which favours effective contraction, and perhaps initiates it. Hence the maximum of health demands the presystolic impulse. A further advantage secured by the auricular musculature arises from the fact that Nature takes the long view, and foresees, and prepares to meet, the impediments which disease may plant in the way of the ventricular filling. It therefore supplies a tissue which, being endowed with the power of growth, enables the overcoming of the hindrance, according to its measure: thus *venienti occurrit morbo.*

These statements apply to both ventricles equally; the differences between the two—and they are great—consisting simply in the difference in quantity of force generated on either side of the septum to meet the mechanical demands presented by each of the two circuits. How can we estimate separately the dynamic values of the two ventricles ?

The most direct and complete way would be

to determine the amount of work done by each systole of the right and left ventricles, but this determination rests upon factors whose individual value is most difficult to ascertain, e.g. in the case of the left ventricle the actual rate at which the blood issues from the heart against an aortic pressure, rising, *pari passu*, as the aorta becomes more and more distended; indeed, everything is in flux—rate of inflow, blood pressure, transformation of current into elastic stretch—and compared with this swirl the ordinary calculations for projectiles would appear mere child's play. Still formulæ have been adopted, and, according to Starling, the relative dynamic values of left and right ventricles so obtained would be about as 5 : 1.[1] The confirmation of these results we must leave to the physicists.

Short of mathematical formulæ, can the problem be approached from any other point of view? A simple method suggests itself in the form of the estimation of the relative amounts of contractile material in each chamber. This might be done theoretically by counting the number of muscular fibres in each; but though the hairs of the head have been numbered, approximately, this method must be discarded as impracticable in the case of the heart, independently of the difficulties introduced by the branching of the fibres.

It then occurred to the writer that results roughly approximate might be obtained with relative ease by the simple procedure of weighing one ventricle against the other, muscle fibre

[1] *Human Physiology*, 3rd ed., pp. 959–961.

preponderating so greatly over every other tissue in the walls of the heart. The argument would then stand: so much muscular fibre, so much contractile force.

Accordingly this method was adopted in a number of experiments which were begun in 1914, but which, unfortunately, were interrupted by the war before they had been sufficiently multiplied. They are given *in extenso* in the sub-chapter, but for reasons about to be given they were not resumed. The method was applied to the auricles as well as the ventricles, and briefly summarized on fourteen cases the results were:

Ratio of both auricles to both ventricles, as 1 : 8·7.

Ratio of right ventricle to left ventricle, as 1 : 2·4.

In comparing the ventricles the whole of the septum was given to the left ventricle, for reasons which have already been given, and which seem conclusive. The small proportion of fibres contributed by the right ventricle to the septum have been regarded as negligible.[1]

Now, though the writer believes that this method is capable of yielding good results, especially on a larger number of observations and an improved, more refined procedure, and that so we should be able to obtain with sufficient accuracy the relative muscularities of the several chambers, he is of opinion that these figures will not correspond with the actual development of force by these chambers: the following are the reasons:

[1] See the appendix to this chapter for further details.

In the first place, because we have no right to assume that all muscular fibres, even of one type of structure—for instance, the cardiac type—have the same power of response to a given stimulus, e.g. that the fibres of the auricles, whose office is to deal with the low pressures obtaining in these antechambers, are capable of the same response as the fibres of the ventricles, which deal with high pressures. The probability is that the muscle fibre may be keyed up or down, so to speak—in fact we know it can.

In the next place, there is as little warrant for assuming that the stimuli supplied by the nerve fibres of auricles and ventricles have the same value; rather would it seem probable that in the case of the former we witness the results of minimal stimulation, in the latter of stimulation which is maximal, the same nerve fibril, structurally, being capable of conveying now a larger amount of stimulus, now a stimulus of higher tension; just as the same electric wire may be used to carry a larger quantity of current or a current of higher voltage.

Thirdly, even granting equality of response of the individual muscle fibre and equality of stimulus conveyed by the individual nerve fibre, still the assumption of equal force output by equal numbers of fibres auricular and ventricular would be unjustified unless we could show that the arrangement of these fibres, their co-ordination, was identical. For just as two systems of levers of higher and lower mechanical advantage would give to the same amount of force working them a greater or less power, so according to the structural arrangement of the fibres of

the several chambers will be their force-output, severally. Can we doubt, looking at the organization of the left ventricle as against that of the right, and of either of these as against that of the auricles, that we have before us structures of a descending order of potency ?

To put it generally : in a given field of forces the effectiveness in one or other direction may be heightened according as we co-ordinate these forces to develop their powers most advantageously.

For these reasons any estimate based on merely relative muscularities must be inadequate, and probably fall far below actualities : hence it has not been thought necessary to extend them further.

In this impasse, between the complexity of the problem presented by the calculation of the work done by the different chambers, and the inadequacy of the estimation of the respective quantities of muscular tissue in their walls, there is a third method which should give reliable data for comparison, viz. by the determination of the blood pressures in the chambers of the heart. For blood pressure is a direct effect of muscular effort and exactly proportionate to it. Strictly speaking, it is static, not dynamic, but given the opportunity it becomes dynamic as momentum, and the quantity of momentum then generated will be directly as the height of the pressure. In the case of the left ventricle the opportunity comes with the opening of the aortic valves, and the intraventricular tension then spends itself in the momentum of the issuing blood stream. The dynamic values, then, as determined from blood

pressure records with the new manometer (i.e. by the optical method) are, in the dog [1]:

For the left ventricle 130–150 mm. Hg (maximal pressures under normal conditions).
For the right ventricle 25–35 mm. Hg (maximal pressures under normal conditions).

This gives a proportion of 5 : 1, approximately, and it will be noted that, in spite of what has been said of the method of calculation by the work done, the results happen to be the same. Be this as it may, the determination by blood pressure alone has the advantage of simplicity.

In the case of the auricles no very definite statements have been come across as to the blood pressure, but we know for certain that, normally, during the greater part of the ventricular diastole it must be below that in the large veins at the root of the neck (for which the B.P. estimation is 0 to −8 mm. Hg [2]), otherwise the blood would not flow into the auricle. The period of maximal pressure, under normal conditions, will come during the auricular systole. Hürthle says of this systole that it generates " a *small rise* of pressure (in the ventricle) . . . lasting about one-twentieth of a second "; if this be so, the pressure in the auricle which generates it can not exceed it by much. We may then take it that in either auricle the maximal pressure must be very small compared with the above figures: the pressure on the left side will slightly exceed that on the right, because, as previously stated, the thicker left ventricle walls will need more force for their filling.

[1] Starling, 3rd ed., p. 945. [2] Op. cit., p. 922.

## THE VALVES OF THE HEART

It is not possible to discuss the forces concerned in the working of the heart without reference to the valves. The auriculo-ventricular valves are, so to speak, the taking-off ground for the systole of the ventricles; it is they which, by forbidding the retrograde movement, compel the forward stress and the forcing of the semilunar valves. In like manner the semilunar valves, upon their closure at the cessation of the systole, compel the elastic recoil of the aorta and pulmonary artery to take effect forwards through the greater and lesser circuits.

The working of the *a–v* valves is sufficiently manifest, but that of the semilunar valves demands some attention. In the first place we must note that the efficiency of these latter is of cardinal importance owing to their position at the very beginning of the two circuits, greater and lesser, where a failure, i.e. of any magnitude, must cause more certain and sudden circulatory disaster than anywhere else: at all costs, then, the efficiency of these valves must be safeguarded. How is this brought about ?

If we look at the aortic valve we see three thin membranes attached at the root of the aorta and so projecting into the lumen of the vessel as to form pockets which open in the direction of the circulation. To the forward movement of the blood the pockets offer no obstruction, but any retrograde movement brings them immediately into apposition and opposition. This is the essential purpose of the valve, viz. to give a forward direction to the *power of the ventricle*

when it has become converted into an elastic stretch of the arterial system. But it does more than this, for, inasmuch as it prevents all reflux into the ventricle, it protects this chamber during the whole period of its relaxation, thus enabling the low pressures in the systemic veins to become effective and the ventricle to receive its full complement of blood. It is clear there must be no hitch in the working of this valve.

Passing now to the act of expulsion of the blood from the ventricle, the question arises, How is it, as these thin membranes are swept aside by the emerging column of blood, that they are not carried too far outwards or even plastered against the walls of the aorta,[1] so that the recoiling blood, on the cessation of systole, should fail to catch their edges and bring the cusps into play? Yet this failure never happens: the closure is unerring.

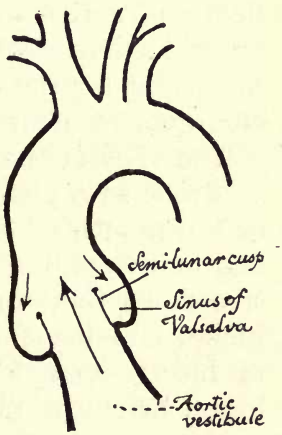


FIG. 12.—Schematic diagram showing the full opening of the aortic valve during systole, as usually accepted.

It was at this stage that the idea occurred whether we had here, perhaps, the secret of the sinuses of Valsalva; for the globular enlargement of the root of the aorta resulting therefrom

[1] This view was at one time seriously held, and the point has even been discussed as to whether the curtains of the valve might not serve to cover and occlude the mouths of the coronary arteries during ventricular systole. Why they should be thus protected was not very obvious.

would render the application of the curtains of the valve to its walls, if feasible at all, a matter of much greater difficulty, and the backward flow of the blood would almost certainly catch in the semilunar pockets and bring the cusps together.

On subsequently looking the matter up, it was found that this very suggestion is made in Piersol's *Anatomy*, ed. 1918, p. 700, and there can be little doubt that this would be the tendency of action of the sinus; but for reasons I am inclined to think that another explanation must be sought for the real significance of the bulbous enlargement of the aorta and of the sinuses.

The view of the ejection of the blood from the ventricle in a piston-like column of the diameter of the mouth of the aorta is entirely imaginary, and in my opinion untenable; for with the arterial tree down to the finest arterioles distended with blood, and beyond them the capillaries also full of blood, even the smallest movement of translation of the whole mass is unthinkable, except upon the view of absolutely rigid vessel walls, since the blood is incompressible. Given such rigidity, the force required would have to be enormous, and it is more than doubtful that any materials could withstand the strain. But the walls of the aorta are not rigid, therefore this hypothesis falls, and in theory the blood might still enter in full column, the artery yielding to receive the excess of blood. To consider this question let us regard the following points:

1. The structure and purpose of the aorta. The first thing which strikes one is the disproportionate size of the aorta to the contents of the ventricle. A chamber of some 4 ounces

capacity requires a draw-off tube of $1\frac{1}{8}$ in. diameter in order to receive its contents! This large calibre requires some explanation.

2. The next thing to observe is the structure of the walls of the aorta, the striking features in which are the great development of elastic tissue, both in the form of fibres and fenestrated membranes, and also the strengthening of the middle coat of the artery by additional connective tissue.[1] These two tissues confer in a special degree elasticity and strength. These are clearly intended to be utilized.

We have seen that in the course of the circulation the intermittent strokes of the ventricle disappear, and are replaced by an even flow through the capillaries and veins. We know that this disappearance is effected in the arterial system, and we now see the *modus operandi*, for the aorta and its main branchings together make an elastic reservoir, the equivalent of the elastic bag which in the hand-spray intervenes between the nozzle and the rubber ball which the hand grasps. As each squeeze propels the air into the bag, and as the act is repeated with sufficient frequence, the bag becomes uniformly distended, and there issues from the nozzle a continuous stream of air. By regulating the squeezes to a certain uniformity of strength and rate, the elastic bag may be maintained at a uniform stretch, and the contained air, therefore, at a uniform degree of compression; and under this constant head of pressure the stream of air from the nozzle will be of even flow. When this stage has been reached each systole (of the grasp) will make

[1] Schäfer, *Essentials of Histology*.

good the loss of elastic stretch due to the escape of gas from the nozzle. The analogy is exact; an intermittent propulsive act has disappeared, and has been replaced by a continuous act of subsidence. Thus likened, the aortic tube becomes of the nature of a transformer which converts an interrupted act into a continuous act. Incidentally this continuity of flow gives the motive force, i.e. muscular contraction, the opportunity to rest and recuperate during the intervals of relaxation. It has been all very carefully planned by Nature.

In the ordinary way we do not speak of a stream of gas *through* the air bag of the hand-spray, but of course there is a flow in the act of subsidence, though it becomes perceptible as current only when we approach the narrowing aperture of outflow. It is the reverse in the case of the aortic sac, inasmuch as the outflow into the capillaries is in the aggregate vastly increased.[1] In respect of the required degree of distension of the air-bag, as this was attained only after many squeezes of the hand ball, so in the course of the development of the organism the blood pressure in the aorta and its branches (otherwise their distension) is represented by the sum of many ventricular systoles; but at any given stage the prevailing pulse rate makes good by each systole for the loss of blood pressure, due to escape through the capillaries, in the course of each cardiac cycle.

[1] Accordingly the subsidence in the aortic sac by its main branchings appears as a current, which is very rapid when compared with the flow through the capillaries. (See also pp. 89, 90.)

We now see the reason for the great size of the aorta and its main branchings, which seems so excessive for the carrying off of the small contents of the ventricle; it is not a draw-off tube, but just a bag *to be kept distended*; its size is necessary to present a sufficient area of elasticity.

It has been said that intermittent action is replaced by continuous action, and so it is in so far as *flow* is concerned; but the intermittent propulsion does not wholly disappear from the scene, for its impact upon the distended arterial system sends a thrill through the whole mass of arterial blood. This thrill appears as a wave whose amplitude and rate of transmission depend upon the force and suddenness of the impact, and upon the tension within the system of tubes and the thickness and elasticity of the vessel walls. This wave is the pulse; it travels right down to the smaller arteries; it has nothing to do with rate of flow.

The necessity for the large calibre of the aorta being apparent, there is no need to explain the size of the aortic valve; it must be large enough to close the aorta completely against regurgitation, for upon its efficiency in this direction depends the maintenance of blood pressure.

But must the whole valve be utilized during systole; does this follow? That is another question, and in its answering we may perhaps arrive at a more satisfactory solution of the purpose of the sinuses of Valsalva and general enlargement of the root of the aorta.

The diagram is designed to show the aortic valve in action just before its forcing by the

intracardiac pressure. It will be seen that the cusps are in apposition between the points *a* and *b*, i.e. from just below the corpus Arantii *a* to the point *b* where the cusps first meet. The line *ab* will correspond with the breadth of the lunula. Here the valve is at its thinnest, but note that this part of the valve, being in accurate contact with its fellow, will bear no strain, for the pressure on either side of this thin spot will be the intra-aortic pressure which will be throughout the same. Everywhere else the intra-aortic pressure is bringing its full force to bear. Above the level *cc* the aortic walls bear the full aortic strain, varying from the maximum blood pressure at the completion of ventricular systole to the minimum B.P. immediately before the aortic valve is forced, the blood pressure having waned during the ventricular diastole. Below the level *cc*, in the aortic vestibule, the blood pressure will vary between the maximal pressure which forces the valve, to the minimal pressure which follows immediately upon relaxation of the ventricle. Note now that the semilunar valves, on their aortic aspect, will be subjected to the prevailing aortic pressure, whatever it be at a given moment, but that over the line *ab* (the breadth of the lunulæ) the cusps will suffer

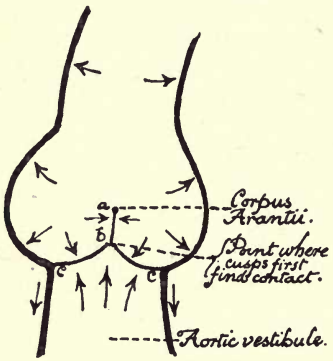


FIG. 13.—Schematic diagram to show the state of the aortic valve just before it is forced; the arrows show the direction of the lines of pressure and of pull.

*no strain* because the pressures on either side give a counterbalancing support: *the tenuity of the lunulæ, therefore, does not impair the strength of the valve.* On the other hand, the cusps between *b* and *c* will be subject to *a strain* which is maximal at the beginning of ventricular diastole, because the lower surface of the valve is then wholly unsupported and the pressure above is maximal.[1] The strain will grow less and less as the intracardiac pressure rises, until it equalizes the pressure in the aorta; there will then be no strain. When the valve is forced and the ejection follows, the cusps thrown outwards and loose in the stream cannot be subjected to any calculable strain; their mode of attachment prevents this.

Let us next consider the actual forcing of the valve. If we look at the preceding diagram, Fig. 13, we see that the intracardiac pressure presents its thrust against the closed valve in the form of a spearhead—actually it will be triangular in shape—each barb fitting into the groove between the cushions of two adjoining cusps; the barb is really a wedge, hollow-ground in cross-section, the convexities of the cushions giving this character to it. In the centre of the aortic valve, at or just below the nodules of Arantius, the

[1] This statement would require qualification supposing that the relaxation of the walls of the heart should follow in a special order, and not all at once, i.e. that the part which contracts first should relax first, that which contracts last should relax last. It has been suggested that there is an order in contraction, and if so, an order in relaxation would be most probable. In that case the terminal contraction at the base might lend its support to the aortic valve at this period of greatest strain on the valve, viz. the beginning of ventricular diastole.

three wedges will unite to form the point of the spear. The whole is a contrivance of great power. Now comes the important question: will the valve yield as a whole or in part? The forces holding the valve against the thrust are everywhere equal, since the tension is everywhere the same above the valve; not so the forces brought to bear on the ventricular side; these converge to the spear point, and here, therefore, the valve must yield first. The inrush will start then as a centrally placed stream; will it remain such? I am inclined to think it will; the grounds for this belief are as follows:

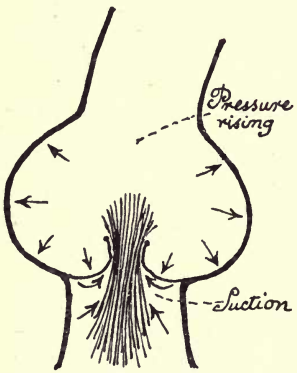


FIG. 14.—Schema to show the hypothetical central current during the forcing of the valve.

1. That a gush of blood occupying the whole calibre of the aortic orifice could not be maintained for the length of the systole; in other words, it would drain the contents of the ventricle too rapidly.

2. That such a column of blood would be too great a strain on the elasticity of the aortic walls, falling as it would in chief part upon the root of the aorta.

3. That the sudden cessation of this current would lead to too violent a reversal of the forces engaged, this being antagonistic to the establishment of an even head of pressure in the aorta—the purposed aim.

On the other hand, in favour of a central stream we have:

1′. That the valve must yield centrally in the first place: why should this yielding not remain localized more or less ? [1]

2′. That, though the moment *before* the forcing of the valve the pressures on the valve above and below are equal, the moment *after* all the pressure relations are disturbed: on the aortic side the pressure is rising rapidly, as the aortic root is forcibly distended; on the ventricular side the pressure is rising also, and in mid-stream is kept at a level just above the aortic pressure, otherwise the grasp on the blood would not be effective. Further, that in the rear of an escaping stream, the current tends to develop a negative pressure; this would suck out the blood from under the valve, or that would be the tendency. I conclude, therefore, that the pressure above, in the pockets of the valve, would preponderate and would maintain the cusps more or less approximated. To attempt to follow more closely the play of forces in the aortic swirl would be wholly beyond the writer's competence.

3′. That a central current, thus limited, would be much more favourable to an equable and gradual distension of the aortic root, through the very fact of its central placing and its more limited volume. From this centre the lines of distension would tend to radiate. I venture to suggest whether we have not here a more reasonable explanation of the globular swelling of this first part of the aorta (including the sinuses of

[1] For it must be remembered that the whole of the valve is held; and that when the central part gives way, the other parts maintain their resistance to any further opening; and, again, that this resistance is a rising one, as is explained in paragraph 2′.

Valsalva), viz. that by presenting thus a larger distensible area, it breaks the first force of the propulsive ventricular act ? The other suggestion made as to the meaning of the sinuses—viz. that they favour the closure of the aortic valve at the end of systole—will be less cogent if the valve is never fully opened, as is now surmised. Closure, indeed, must follow inevitably without further aid, on this supposition, and the disturbing effects of the recoil would also be greatly minimized by the smallness of the area of recoil.

But, it will be said, after all this is only surmise, have you any proof that such a limited inflow would be capable of maintaining the aorta adequately distended and therewith an effective circulation ? Can you show that a *central* flow has ever existed or coexisted with full health ? To both these questions the answer is, yes.

It is pathology which brings important evidence on these points, and first as to the *volume of inflow* necessary to sustain life and health. If we look up the cases of chronic disease of the aortic valve, characterized by fibrous and calcific changes, which Dr. Peacock cites in his Croonian Lectures for 1865, we find striking instances of deformity of the valve, with marked thickening and induration of the segments, in addition to calcific deposits, as a rule. In these cases two segments of the valve would at times be found fused into one, at other times all three segments might be thus fused, and in most there would be marked obstruction, sometimes extreme.

The figure shown is taken from Peacock.[1] The

[1] *Valvular Disease of the Heart*, op. cit., pp. 22, 23 et seq.

patient was a woman in the Royal Free Hospital, seventy-six years of age, who had enjoyed good health throughout her life. There was no rheumatic history nor any record of heart symptoms. The patient was admitted for a strangulated hernia and died shortly after the operation. Up to the time of her admission she was in her usual health and able to mount to the top of her house without difficulty.

The valve was enormously thickened and very rigid. Two of the segments were fused into one, and the aperture reduced "to a mere slit 10 lines in length"; this slit could only be opened "to the extent of 3 or 4 lines." Dr. Peacock goes on to cite a similar case recorded by Corvisart, also a woman and of seventy-six years. She had suffered from heart symptoms from her sixty-seventh year onwards. Again the orifice was reduced "to a mere chink," which could only be opened to the extent of 1–2 lines (the mitral valves were also thickened and rigid). The symptoms in this case did not appear until the last nine years, yet the type of change was such as is generally held to be of many years' duration. Moreover, in this case, the mitral valve was seriously affected, so that, all things considered, the crippled aortic valve had not served her badly.

Calcified aortic valve.

FIG. 15.

Two cases, even more striking, are quoted from Dr. Stokes[1]; in the one case the patient

[1] *Diseases of the Heart and Aorta.* Unfortunately, in these cases and in those of Dr. Peacock's own observation there is not

*was a man of middle age who up to a few days before death* "had enjoyed uninterrupted good health," yet the aortic valve was reduced to a very small slit about 4 lines in length, through which it was just possible to pass a fine probe. The other case was a man of fifty-four years, who was *free from symptoms till six months before his death*; he was seized whilst walking up a hill; this was his initial symptom. The aortic valve would only admit "the passage of a fine quill."

In all these cases we must make allowance, in respect of the measurements, for the fact that the vessels were not under tension [1]; but allowing in full for this, there is no question about the extreme limitation of the volume of the stream —yet life attains to the mid-term or even to the full term and beyond, either without symptoms, or with their development deferred until near to the final event; and this in the presence of changes which in their extreme form will have been in existence for years, probably many years.

There is a beautiful specimen of Hunter's in the Royal College of Surgeons' Museum, now numbered 645·1, showing the same kind of change

sufficient reference to the state of the left ventricle as to hypertrophy, though on p. 23 of Peacock's work we find the general statement: "In cases of this kind there can be no doubt the changes in the valves must have been slowly progressing for many years, the obstruction occasionally being compensated by the increasing power of the ventricle, so that no symptoms of disease were caused."

[1] In Dr. Stokes's first case (referred to by Peacock, op. cit., p. 23) it seemed, on first inspection of the valve from above, that there was no opening at all, but from below a "very small slit was discernible." Under the intraventricular systolic pressure the slit would have been much more demonstrable.

in a pronounced degree. The opening into the aorta is reduced to a narrow fissure. It came from the body of a man "who had been very strong, but was greatly emaciated"; but details as to age and the presence of symptoms are lacking.

The next case is also figured from Peacock.[1] The interest lies in the central placing of the aperture. The disease has proceeded very symmetrically towards the centre, leaving a gap just such as would be made by the forcing of a normal valve at its centre by a triangular-shaped spearhead, as suggested in the foregoing argument. The patient was a man of forty years, "who had been ill for three weeks when I [Dr. Peacock] first saw him." The illness was of an acute nature and was ascribed to chill; it followed a rapid course, and a week subsequently he died. This was in 1857, when temperatures were little taken. What the nature of the affection was it is impossible to say; the patient came from a district in which ague "was very prevalent at the time." Death occurred with symptoms of acute heart failure. It will be noted that in this case we have regurgitation as well as obstruction, and without doubt the former will have been the more lethal lesion; but the point to accentuate is that the patient "had never been observed by

Calcified aortic valve

FIG. 16.

[1] Op. cit., p. 18.

his friends to be short-breathed on exertion. He was somewhat irregular in his habits, had been addicted to hunting and horse exercise, and had never had rheumatism or any other serious disease." Yet this was the valve which served his irregular habits and physically active life, without occasioning any distress or other symptoms.

These cases answer definitely the questions propounded, for they show in the first place that a very limited inflow of blood into the aorta, as compared with the volume of the stream which the aortic orifice could convey, if fully utilized, does answer all the needs of the circulation; and next we have evidence in one very striking case of the existence of a central stream of very limited area, such as that suggested in the foregoing argument, which also furnishes an effective circulation.[1] Cases such as these are incredibly difficult of interpretation if it be assumed that the whole area of the aortic orifice is utilized by the ventricular systole, and necessarily utilized, in order to obtain a normal circulation; the difficulty vanishes if we assume that, in fact, the systole makes use of a limited area only of the valve. On the other hand, as has been insisted, the whole area of the valve is utilized in the recoil; this being essential to the maintenance of a due blood pressure and the ventricular refilling.

It does not follow that the area of the valve actually used in health is *as limited* as these cases of disease show, for we have the evidence of hypertrophy to show that in these latter

[1] See also Case VII, pp. 19, 20, Peacock, op. cit. The patient here was a man of forty, and all three aortic cusps were extensively diseased and blended.

an extra effort was required to overcome the obstruction.

To approach this problem from another point of view, viz. that of general pathology, we note that when disease enters upon the scene, its spread from the focus of origin seems very often to be haphazard and unaccountable; but when we observe that a particular line of spread tends to recur again and again, we assume with reason that a definite cause is present. Now in these cases of chronic aortic valve disease a very common line of spread is from the periphery centrally, whereby the adjacent sides of the cusps are apt to be fused into one. All three cusps may suffer thus, and the result is then such as we see in Fig. 16; or only two cusps may be affected, and then we get the conversion, more or less complete, of two cusps into one, as seen in Fig. 15. This blending is often accompanied by a partial destruction of the fused portions; at times this is so thorough that there is little trace of the mode of origin of the single large valve. These facts come out again and again in Dr. Peacock's treatise; and in like manner, if we study the specimens on the shelves of the museums, we find them amply exemplified. Why is this? The answer here suggested is—because the cusps of the valve present lines of contact which are more or less maintained. But, it will be urged, these contacts are constantly interrupted as the segments are flung back, 50, 60, 70, or 80 times in each minute, by the systolic inrush. This is indeed a valid objection, if the separation of the segments each time is up to

their very points of insertion in the aortic wall, for the spread of a chronic fibroid change and the fusion of two surfaces is very difficult to imagine under such conditions of physical interruption. The conclusion which these lines of spread suggest is that contact between the adjacent cusps is not wholly broken but largely maintained throughout the ventricular systole.

Accepting the forcing of the aortic valve at its centre as actual, it is impossible to say how far

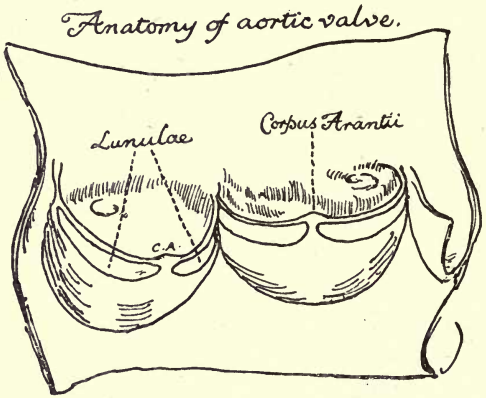


FIG. 17.

outwards it extends; just as it is impossible to say how far into the aortic tube the movement of translation (the current) is propagated before it is wholly extinguished by the elastic force of the aortic walls, into which it has by that time become converted. These matters will vary in each individual, and in the same individual at different times, according as the vigour of the systole varies.

The accompanying figure is taken from Morris's *Treatise on Anatomy*, 4th edition, 1907, p. 486. It shows well the strongly defined fibrous edge

of each cusp reinforced as to its centre in the corpus Arantii. Immediately below the edge the crescents of the lunulæ are seen: this is the most fragile part of the cusps; but it will be remembered that, when the valve is in action, these parts are in close apposition with those of neighbouring cusps, the closer because of the delicacy of the membrane of the lunulæ; and that, being in contact each with the other, they get mutual support and hence do not feel the strain of the blood pressure. Immediately below them the valve is again reinforced by bands of fibrous tissue which are required because these parts of the cusps, receding from each other, have to bear unaided the force of the aortic recoil. The free edge of the cusp is strengthened by a special tendinous band, and in like manner its convex border of attachment to the wall of the aorta is similarly strengthened. From the last-named the interlacing fibres which traverse the main body of the cusp have their general direction towards the nodule of Arantius.[1]

The aortic valve in its three parts appears thus to secure (1) firmness of the lines of attachment; (2) security of the vulnerable free borders; (3) accuracy of contact of the lines of apposition (by means of the thin lunulæ).

The sinuses of Valsalva are well defined in the figure.

Does any special function belong to the corpora Arantii? Piersol[2] suggests that they serve to fill the gap left at the central meeting-point of the edges of the three cusps, but it does not seem very clear that this would be secured by

[1] Morris's *Anatomy*, loc. cit. [2] Loc. cit.

the junction of three nodules; indeed, the accuracy of contact of an edge which is bent at a given point would seem rather to be hindered by thickening the edge at that point. Is it not more likely that the significance of these bodies is to give structural strength, as concentring the fibres which make up the substance of each membranous segment?

## SUB-CHAPTER

### EXPERIMENTAL FINDINGS

The total output of energy demanded by the act of the circulation of the blood is the sum of the energies developed by all the four chambers of the heart. Each chamber contributes its share of momentum, and according to the magnitude of the contribution, so is the order of their placing. Judged thus, and in a purely mechanical act a purely mechanical estimate forms the only criterion, the left ventricle stands *facile princeps*, its effort far exceeding the combined efforts of the other three chambers. The right ventricle comes next, and then, at a long interval, the two auricles. These last-named yield relatively such small quantities of momentum, that they can rank only as subsidiary, almost negligible auxiliaries to the ventricles. Compared one with the other, the two auricles must come very close, but the left auricle should put forth the larger effort since it has to distend the thicker walls of the left ventricle.

These statements are very general, and it seemed desirable to make them more specific if possible—the question was how? In each chamber the energy-developing tissue is one and the same, the muscular fibre; there is no other source of energy engaged in the movement of the blood. It seemed, therefore, at first sight, that if one could determine the amount of muscular

fibre allotted to each chamber, a simple answer would be forthcoming. To this end two methods presented themselves: (1) to estimate the quantity of muscular tissue by volume; (2) to estimate it by weight. Of these two, the former did not appear feasible, whether the attempt were made to get at volume by displacement-of-fluid methods or, still more difficult, by the enumeration of the heart fibres, this last involving the primary assumption that all fibres were of one size. Much more simple, and promising results of approximately definite value, seemed the estimation by weighings, and accordingly this was adopted.

Method of procedure:

Specimens from the post-mortem room, fresh or preserved, were selected, for since we were dealing with relative values, the effect of preserving fluids upon the fibres might be discounted since, presumably, this effect, in any given case, would be identical upon the muscular fibres of each chamber, and the relation therefore undisturbed. As far as possible, all external subpericardial fat was removed by dissection (hearts varied enormously in this respect); it was not possible to deal with interstitial fat. As to the pericardial covering, this was not touched except in so far as it was removed with the subpericardial fat.

The auricles were then carefully separated from the ventricles and were kept in one piece, no endeavour being made to estimate one auricle against the other. The reason for this was that in health, judging by the naked eye, the two auricles appear to be nearly equal, and, further, that the septum is in all probability equally shared; there seemed, therefore, no object in attempting the difficult and arbitrary task of dividing up the septum.

The ventricles came next, and here the question of the septum became matter for serious consideration—how, namely, to apportion it between the two. As the investigation proceeded, it became more and more clear that for all practical purposes the septum must

be assigned *in toto* to the left ventricle. That some longitudinal fibres proceeding from the apex of the right ventricle to the base (including some few fibres supplying occasional, irregular, and supplemental chordæ tendineæ attaching the tricuspid valve to the septum) belonged to the right ventricle was certain, but they formed a negligible proportion of the mass of the septum. The cross-sections of the heart in systole or in diastole were most convincing as to the validity of this assignment, the septum carrying on and completing the curve of the other parts of the left ventricular wall, so as to form a circle whilst maintaining an equal thickness with those other parts. Thus by means of the septum, wholly appropriated, the left chamber becomes fitted for concentric contraction, and at the same time for the development of uniform power at every part of the contracting circle. Contrasting with this the D or crescent shape of the cross-section of the right ventricle, we perceived nothing in the line of the septum which conformed to the line of the other parts of the ventricular wall so as to associate the two parts in common concentric contraction. Moreover, it was not possible to conceive of the presence of transverse fibres in the septum, some of which should belong to the left ventricle, and some to the right, for these entering into simultaneous contraction in the ventricular systole must conflict, mutually paralysing the effort of either chamber, and tending at the same time to disrupt the integrity of the septum.

Accordingly, after the first few observations, in which the septum was separately removed and weighed, it was always treated as an integral part of the left ventricle. The right ventricle was divided from the left by incisions which kept the lines of the interventricular sulci, S and S′, but were carried obliquely so as to follow the curve of the left ventricle, i.e. by the tangential lines AB, CD. (See Fig. 18.)

To explain now the method of calculation, it will be

simplest to take a given case, that of a patient named E. H.:

| | grammes |
|---|---|
| The weight of the combined auricles and septum was . | 33 |
| The weight of the combined ventricles and septum was | 168 |
| The weight of the right ventricle was . . . . | 43 |
| The weight of the left ventricle and septum was . . | 124 |

Now the relations of these figures, as they stand, would not give correctly the dynamic values if we compare auricles with ventricles, for whereas we may reckon for the auricles and septum, with their thin layer of loose-meshed muscular fibres, that at least 50 per cent. would consist of fibrous covering and lining with interstitial areolar tissue—in the case of the ventricles and their septum the fibrous coverings, even with the interstitial areolar tissue, would probably not exceed 5 per cent. to 10 per cent. of the weight; so predominant is the muscular element. In the table which follows, 50 per cent. was allowed for the fibrous structures of the auricles and their septum, and 10 per cent. for the same structures in the case of the ventricles and their septum. The allowance is an arbitrary one and may call for considerable correction, but it would probably understate rather than overstate the muscular superiority of the ventricles.

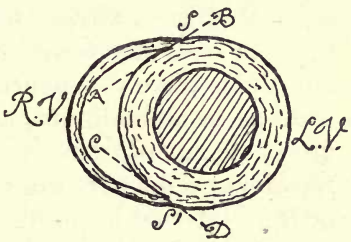

FIG. 18.

| | grammes |
|---|---|
| Making these corrections, we should have for the muscular value of the auricles and septum (33 ÷ 2) . . . . | 16·5 |
| And for that of the ventricles and septum 168 — 16·8 grammes | 151·2 |

∴ the dynamic values of *auricles to ventricles* would stand at 16·5 : 151·2, say, very roughly, as 1 : 10

In the case of the ventricles it was not thought wise to attempt even a rough estimate of the comparative amounts of fibrous tissue, which, of course, will work out adversely to the true muscular preponderance of

the left ventricle, since relatively there must be more fibrous tissue, as compared with muscular fibre, in the right ventricle than in the left, the walls being so much thinner, whilst the pericardial and endocardial coverings will be about the same. Accepting this shortcoming, the muscular, i.e. dynamic, values of the right ventricle and left ventricle will be as 43 : 124 : : 1 : 3 approximately.

In the table which follows it has not been thought necessary to give the actual figures of the weighings; these were obtained in each case in exactly the same way, and the calculation made precisely as shown above, with the deductions for the fibrous structures in the case of the relation between auricles and ventricles.

In the list on p. 83 Nos. I, IV, V, VII, VIII, X showed more or less evidence of left ventricular hypertrophy associated either with evidence of kidney disease (uræmia) or with valvular heart disease; in one of the cases, No. VIII, there was pontine hæmorrhage, but no statement as to albuminuria, and the kidneys did not show gross evidence of sclerosis. Excluding these six cases, the remaining eight were cases either of death from accident or from internal hæmorrhage, or after operation, and in none is the heart mentioned as diseased. These eight cases give an average of 1 : 8·7 for the relative muscularities of auricles and ventricles, and of 1 : 2·4 for the relation of the right ventricle to the left ventricle. We may regard these eight cases as examples of normal hearts, but of course, to get a representative average, the numbers would have to be considerably multiplied.

It was the author's intention to extend these observations considerably, but they were interrupted during the war by daily attendance at a military hospital, and could not have been resumed until 1920. That they were not resumed then was for reasons which will now be repeated.

As they stand what can be said of them? It will be best, perhaps, to sum up the question in a series of propositions as follows:

TABLE OF RELATIVE DYNAMIC VALUES OF AURICLES TO VENTRICLES, AND OF RIGHT VENTRICLE TO LEFT VENTRICLE

| — | Auricles to Ventricles. | Right Ventricle to Left Ventricle. | Remarks. |
|---|---|---|---|
| I. Elizabeth Harris, *æt.* 38 | 1 : 10 | 1 : 2·9 | Spirit preparation from a case of death from uræmia: moderate left ventricular hypertrophy. |
| II. Maud Badman, *æt.* 56 | 1 : 8·5 | 1 : 2·6 | Spirit preparation for case of death from shock due to injury. No signs of valve disease; heart much overlaid with fat and complete removal of this difficult. |
| III. Edward Drummond, *æt.* 34 | 1 : 10 | 1 : 2·45 | Spirit preparation. Death from perforated duodenal ulcer. General appearance of heart, valves, and muscle, healthy. |
| IV. Elizabeth Hilson, *æt.* 26 | 1 : 10 | 1 : 4·3 | Cor bovinum, a striking instance of advanced disease of aortic valves (vegetative, i.e. ulcerative); other valves healthy. Sudden death. Double aortic bruit and mitral systolic. The septum in this case was markedly convex towards the right ventricle. |
| V. Joseph Thompson, *æt.* 60 | 1 : 10·6 | 1 : 2·9 | Spirit preparation. Case of uræmia. Granular kidneys. Valves normal; left ventricular decidedly hypertrophied. |
| VI. Louisa Ward, *æt.* 39 | 1 : 6 | 1 : 2·3 | Chronic alcoholism. Hæmatemesis. Heart small; muscle flabby, pale. |
| VII. Sarah Perkins, *æt.* 48 | 1 : 6 | 1 : 3·26 | Case of "acute abdomen" with pancreatic tumour; operation impossible because of marked heart failure. P.M. mitral stenosis; aortic valve cusps thickened. |
| VIII. Sarah Hoare, *æt.* 48 | 1 : 11·8 | 1 : 3·7 | Pontine hæmorrhage. Death by coma. Illness about sixty hours' duration. Left ventricle a good deal hypertrophied, much superficial fat. Kidneys not shrunken or fibroid. |
| IX. Edward Rushton, *æt.* 10 | 1 : 9 | 1 : 2·4 | Operation case for appendix—development of subphrenic abscess and subsequent empyema. Heart valves normal. |
| X. Edwin Colgate, *æt.* 49 | 1 : 9·5 | 1 : 3·7 | Case of actinomycosis; collections of pus free and encysted in peritoneal sac. Aortic valves very atheromatous, two segments fused, but doubtful if any appreciable obstruction or incompetence. Left ventricle hypertrophied. |
| XI. Frederick Fraser, *æt.* 51 | 1 : 9 | 1 : 2·5 | Operation for primary sarcoma of axillary gland. Post-operative development of infection. |
| XII. Arthur Fryer, *æt.* 8 | 1 : 12·8 | 1 : 2·5 | Lift accident; depressed fracture of skull. |
| XIII. Thomas Cahill, *æt.* 36 | 1 : 12 | 1 : 2·15 | Death from fractured ribs; surgical emphysema; dyspnœa. Heart normal. |
| XIV. D. Timony, *æt.* 39 | 1 : 6·2 | 1 : 2·4 | Intestinal obstruction, chronic; several operations; death after colotomy. |

1. The method is based upon the assumption that the dynamic values of the several chambers of the heart must stand in direct proportion to the quantity of force-generating tissue in each. This must surely be admitted, and therefore, in principle, the method is sound.

2. Though sound in principle, it must be allowed that in performance it admits of much more exact execution, as by a more accurate isolation of the muscular fibres from their framework and its contained fat, whether by more careful dissection or by means of solvents; the figures obtained must therefore be regarded as only roughly approximate.

3. That to obtain the physiological power-values by this method it would be necessary to confine the observations to normal specimens; deviations from the normal would call for contrasting observations on a series of pathological preparations.

4. But that, however perfectly executed, this method can only carry us up to a certain point, it cannot establish the full dynamic values, because other factors, besides mere quantity of force-*generating* tissue, enter into the problem. The point then which we shall have reached by the above method will be that dynamic values are directly proportional to quantity of muscular fibre as *one* factor in the problem.

5. That of other unrepresented factors, one will be the degree of stimulation which is brought to bear upon the muscular fibres through the nerves, i.e. the force-*liberating* structures; for we have no right to assume that the amount of stimulation, whether in quantity or in intensity (the analogue of voltage in electrical phenomena), is the same per fibre when we compare the sluggishly contracting auricle with the tensely contracting ventricle, indeed such equality of stimulation would seem to be very unlikely.

6. That another factor concerned is the structural relationship of the muscular fibres, of which mere

quantity or weight takes no account. Thus it must be apparent that the fibres can be arranged so as to co-operate more or less effectively with their neighbours; and as this obtains, so will their individual efforts, by summation, develop a larger or smaller output. Inspection of the muscular arrangement of the fibres in the auricles and ventricles respectively will leave little doubt as to which structure will give the more effective combination.

7. Accordingly we may formularize the problem thus: D.-V. $\propto$ Q.S.C., i.e. Dynamic value is directly proportional to the Quantity of muscle fibre, to the Stimulation of that tissue and to the Co-operative value of its structure; these three factors multiplied together will give the full dynamic value.

8. But there are no means available at present for gauging the degree of stimulation conveyed by the nerves, nor the mechanical advantage which structure confers, and therefore the estimation of the dynamic values by quantity of muscle tissue is quite inadequate, though in the right direction: the figures obtained will consequently much understate the real values.

9. In these circumstances the only sufficient solution which offers is the determination of the actual work done by each chamber in the act of contraction, a problem of many factors, or, failing this, the determination of the maximum blood pressure developed in each chamber. The last-named is the simpler procedure, and it may be described as a static solution in contrast to the dynamic solution by calculation of the work done.

Weighing all these considerations, it was thought unnecessary to multiply the observations already made.

## CHAPTER III

THE CIRCULATION OF THE BLOOD, GENERAL CONSIDERATIONS WITH SCHEMA — SIGNIFICANCE OF THE ANASTOMOTIC VESSELS—UTILIZATION OF THE BLOOD AS A CARRIER OF REMEDIES—OBSTRUCTION IN ARTERIES AND VEINS; ALSO IN THE HEART CHAMBERS—BACK-PRESSURE EFFECTS.

HAVING dealt with certain structural features of the heart and its main vessel, the aorta, and having considered their interpretation in terms of function, let us turn to the mechanism as a whole and regard its performance. The performance of the heart is the circulation of the blood, that is its aim and end, and, as it achieves this, successfully or otherwise, so will it, the heart, be judged. It is not then to the purpose to say this heart is acting irregularly; the valves of that heart obstruct where they should give free passage, or yield where they should hold; the mechanism therefore, in either case, is faulty, and consequently inefficient. Not thus must we conclude. The true criterion to be applied is—how is the blood being circulated, in actual fact? For, though the data obtained by investigation, coupled with the experience gained from past observation, may enable us to look ahead and foresee, more or less accurately, a coming incompetence, and perhaps ward off or delay its advent; we must always remember that, after all, this is

surmise, which rests largely upon factors very difficult to determine, such as the state of the heart muscle and its reserve powers—the integrity of the nervous supply and its potential (its voltage, if one may borrow an analogue)—the stationary or progressive nature of the defect present. Consequently we must always come back from surmise to the solid ground of present actual performance and review our findings in its light.

The circulation presents us with the pulse, and from its palpation we learn whether the heart is acting regularly or not, in respect of time-sequence and force; whether the vessel is well or ill filled; at high or low tension; also whether the heart's contraction develops and subsides gradually, i.e. is well sustained, or is ill sustained, i.e. is abrupt both in onset and decline—the latter giving us the short sudden pulse as contrasted with the pulse of measured rise and fall.[1] All these observations are immediately referable back to the heart. Certain other observations of the pulse are of purely local significance, such as the suppleness or rigidity and thickened condition of the walls; the state of the channel, free or encroached upon by the thickening. But again these data must be controlled by the actual flow of the blood stream, as it declares itself in the warmth of the extremities and superficial parts; the colour of skin and mucous membrane; the presence or absence of areas of stagnation and tumefaction, superficial or deep; above all, by the evidence of the functional activity of the organs generally, whether vigorous or impaired,

[1] These latter qualities of the pulse will depend much upon the arterial tone.

according as they are well or ill served by the circulation.

It must be added that the pulse presents other features, which require instrumental methods for their determination; but the curves so obtained and the inferences deduced therefrom must again be brought back to, and controlled by, the test of actual circulatory performance.

The composition of the blood may be, of course, a matter of cardinal importance, but it hardly comes into consideration here, for we are dealing simply with the power of the heart to circulate the fluid, such as it is, which the tissues place at its disposal: it is not responsible for the composition of the blood, except in one respect, namely, its aeration good or bad; herein we may have direct evidence of circulatory competence or failure.

The scheme of the circulation is simple: the greater, or systemic, circuit shows us a reservoir of blood under high pressure (the aortic sac); a reservoir of low pressure (the great venous sinuses presented by the large capacity of the main venous trunks and their terminal expansion into the right auricle); an extensive area of canalization consisting of a network of minute intersecting channels (the capillary system); vessels leading from the high-pressure reservoir to the capillaries (the arteries); vessels leading from the capillaries to the low-pressure reservoir (the veins); a pumping apparatus to maintain the high-pressure level in the aortic sac (the left ventricle); another pumping apparatus to return the blood from right auricle to left auricle,

thus completing the circuit (the right ventricle)—incidentally this last act carries the blood through the canalization area of the lungs, thereby aerating it. This smaller trajectory through the lungs is called the pulmonary or lesser circuit; it is really only part of the whole circuit and is complementary to the so-called greater circuit, itself incomplete: the full round of the circulation includes both circuits. The presence of valves in the right and left ventricles enables these power-stations to develop the requisite blood pressures.

Through a system of vessels so disposed there must be flow, so long as the ventricles continue to generate force—flow in one direction always. The generation of force is proportional to the resistance which the system of tubes opposes to the flow of the blood, and this resistance varies greatly in different parts of the system: thus it preponderates enormously in the capillary area, where the blood mass is broken up into fine hair-like streams, and where in consequence the area of surface friction, offered by the channels through which the blood threads its way, is correspondingly increased. Accordingly, of the 100 mm. Hg pressure, which is averaged in the aorta, the chief part is spent in overcoming the capillary resistance. It has been said that the flow through the blood vessels is constant in direction; this is true, but in rate it varies, as does the resistance, enormously. The explanation of this is to be found in the vast disparity between the areas of cross-section of the system of tubes at successive points of the circuit.

How this disparity works will appear from the simplest of calculations, for if two points be

selected—the one, say, at the aorta in its first part, and the other in the capillary area—and we imagine a cross-section taken at each of these two points, then, assuming for argument's sake that the area of *cross-section* through the capillaries is one hundred times as great as that through the aortic trunk, the *velocity* of the current at the aortic section must be exactly one hundred times as great as that at the capillary section, since the same volume of blood must pass through either cross-section in the unit of time: on no other terms could continuity of flow be maintained, and continuity there is. Of course, no such cross-section through the capillaries is anatomically feasible, for by "capillary area" we mean the sum of all the capillary areas of the organs and tissues fed by the systemic circuit; but this does not touch the force of the argument or the strictness of its conclusion, viz. that the rate of flow at given points of a circuit of ramifying tubes is inversely as the cross-sectional areas at those points. Now physiologists have calculated from other determinations that the rate of the capillary flow is 0·5 mm. per second, whilst the maximal rate in the aorta is 500 mm. per second, i.e. 1,000 times as great[1]—it follows that the capillary cross-sectional area is 1,000 times as great as that of the aorta. What do we deduce from this, physiologically? Why, that the business of the blood is performed in the capillaries, that is, at the place where it spends its chief time. From the heart the blood is hurried as quickly as possible along the arteries

[1] Starling, op. cit., p. 931. Should these figures subsequently call for correction, this will be a question of detail only.

in order to reach the capillaries, and, having passed through these, it is gathered again and returned as quickly as may be to the heart: arteries and veins are merely lines of communication, in which the metabolic processes are reduced to a minimum.

We need not delay over the varying velocities in arteries and veins at different points of the circuit, but it is a matter of some interest and practical application to inquire as to the time taken by the blood to complete the whole circuit. Specifically the question to be put is—how long will it take for the contents of the left ventricle, in the act of crossing the aortic valve, to return to the same point? The trajectory will include, obviously, both the greater and the lesser circuits. At first sight the problem may appear to be very complicated and to involve a very nice determination of the several velocities at different points, as well as of the lengths of vessel over which it might be assumed that these velocities would be practically constant; then would follow a summation of results. Such a problem would, in fact, be almost incalculable; but all these intricacies may be short-circuited in a very simple way; at least that is the suggestion now to be proposed for consideration.

At each cardiac cycle the contents of the left ventricle are propelled into the aorta, let us say 4 oz.[1] At the completion of any given systole,

[1] Henle says the estimations of this quantity vary between 60 and 150 c.c. (*Gefässlehre*, 1876, p. 45); the middle term of these figures will be 105 c.c., just over 3·5 oz. In Cunningham's *Anatomy*, edited by Arthur Robinson, 1915, the capacity of all the cavities of the heart is given as about 4 oz. each (see p. 880).

this quantity will find itself rearmost in a queue of many antecedent systoles. Within the system of tubes larger and smaller this position will be maintained practically, for there will be no appreciable mingling except at the very outset, before the aortic swirl has disappeared and all movement of translation been converted into elastic stretch. From that time onwards the blood last injected will keep its position in shorter and shorter column as the arterial branchings multiply, until it is spread out in a thin line in the capillary network. Here, it is true, diffusion may take place, but that may be disregarded, for the whole is in forward movement, and the thin line may be taken as still rearmost. In the veins the thin line will be gathered together as the channel narrows, and in the end, near the heart, the blood will be again in column-formation, and so it will enter the auricle and right ventricle. The process will now be repeated through the lesser circuit, and finally the left ventricle will be reached and the whole circuit accomplished. Regarded thus the entire volume of the circulation will be composed of a series of 4-oz. volumes in line, which in fact it is, and reckoning the total blood mass at one-twentieth of the body weight,[1] and the latter at, say, 160 lb., then with a pulse rate of 60 per minute, a very simple calculation gives us the

[1] Starling, op. cit., p. 897. Since this was written it appears that a still more recent estimate puts the blood mass at one-thirteenth of the body weight (Lyle and de Souza, 1921). This brings us back to the teaching of the writer's student days, and would of course lengthen the duration of the circuit. These divergent estimates do not touch the validity of the *method* here advocated.

number of this series, viz. $\frac{160}{20}$ = 8 lb. (the blood weight); this in ounces = 8 × 16 = 128; and this divided by 4 (the ventricular capacity) = $\frac{128}{4}$ = 32 systole-volumes, each of which succeeds the other second by second. ∴ the double circuit will be completed in 32 seconds.

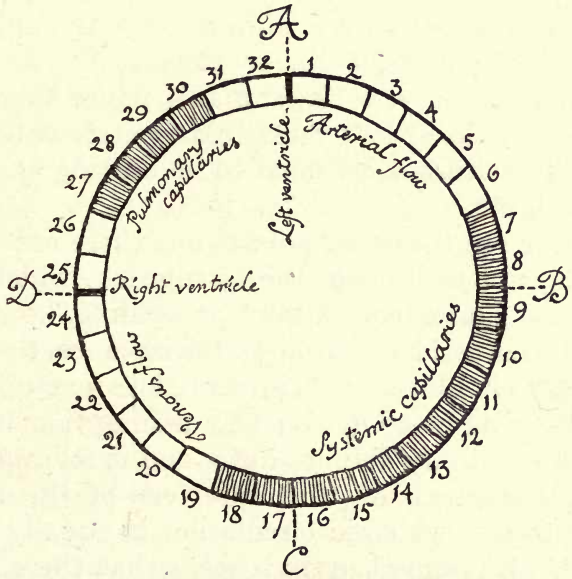


FIG. 19.—Schema to illustrate the foregoing calculation of the time necessary to complete the blood circuit and other time relations.

The figure is entirely diagrammatic: concerning it we must note that:

1. The total blood volume is shown as contained in a circular tube of uniform bore.

2. This blood volume is broken up into thirty-two equal parts, each of which represents the ventricular capacity (this we may take by common consent to be the same for either ventricle).

3. The actual figure is calculated upon a body weight of 160 lb.; of which weight the one-twentieth part, i.e. 8 lb., is assigned by physiologists to the blood mass.

4. It is calculated also upon an average ventricular capacity of 4 oz., which figure is about a mid-term between the extremes of ventricular capacity, as stated by Henle, viz. 60 c.c.—150 c.c., or 2 oz.—5·33 oz.

5. The number 32 is obtained by dividing the total blood mass, 8 lb. or 128 oz., by the ventricular capacity, 4 oz., and this figure therefore stands for thirty-two ventricular systole-volumes.

6. The heart is assumed to be beating at sixty per minute.

These are the main points, but there are other points, not utilized in the calculation, which the diagram illustrates without pretending, however, to any numerical accuracy; for instance, the pulmonary circulation is represented as one-quarter of the total blood mass; this assumption is not based upon any definite data, and it follows that the assignment of three-quarters of the blood mass to the systemic circulation is equally arbitrary. But the diagram teaches that there must be a great disparity between the blood volumes in the lesser and greater circuits; that is a truth.

In like manner the shaded parts assigned to the capillary circulations in either circuit are quite arbitrary, numerically; but they indicate the fact that the greater part of the circulation period will be spent in the capillaries, where the obstruction to the flow is greatest; here the true business of the circulation is transacted, for the heart does not chase the blood round and round

for the sake of making it run. The actual rate of movement through the capillaries, it will be remembered, is put at 0·5 mm. per second. It is probable that the rate differs for the two sets of capillaries, and that, through the pulmonary capillaries, which are wider, there is a quicker movement, though *per contra* the motive power is, of course, much less. On all these points the physiologists must come to our aid, it is they who must test not only the correctness of the method, but must also be responsible for the accuracy of the data.

The unshaded segments in both circuits represent the blood volumes in the arteries and veins ; they are arbitrarily set down as equal, as are also the periods which those volumes represent (each segment a second) ; this would mean that the velocities were the same. On both counts these assumptions are unwarranted and almost certainly incorrect ; but both counts refer to non-essentials and unimportant factors, which have no concern whatever with the method of calculation of the *whole period* of the blood circuit as here presented ; and it would have complicated the diagram, without serving any useful purpose, to have attempted to differentiate these.

In the diagram the right and left ventricles are represented as separate, for, though simultaneous in action, and organically attached the one to the other, they are in effect sundered by the septum and are placed at different points of the circuit, as shown, viz. the one at the entrance into the systemic circulation, the other at the entrance into the pulmonary circulation : they are in reality two distinct hearts.

It may anticipate objections as to the method of calculation if we admit at once that, since the body weight varies, the blood mass in exact proportion must vary also; and so by keeping the same divisor, "4," which here has been set as the ventricular capacity, we should get markedly different estimates for the blood circuit time. Of course we should; and therefore the divisor must vary in the same proportion as the blood mass. And surely this will be so, for the maintenance of a uniform mean blood pressure in the aorta must depend exactly upon the maintenance of a uniform *proportion* between the capacity of the left ventricle and the capacity of the vascular circuit which it serves: the same, of course, will hold in respect of the right ventricle and its circuit. This proposition should hold for healthy individuals: disease local or general, including obesity, would be almost sure to disturb it more or less.

To conclude—this method of measuring the duration of the whole period of the blood circuit rests on the assumption that at any and every given point of the vascular system there passes through the cross-section at that point the same volume of blood in the unit of time. At two points of the circuit we have direct proof of this, for in the same unit of time there passes the same volume of blood through the right and left ventricles respectively; their simultaneous action and equal capacity declare this.

How is this remarkable uniformity of result achieved?

The vascular system as a whole is made up of many subsidiary systems; in fact, as many

organs and tissues as there are, so many are the vascular areas; and each one of these is vulnerable, and one and all are exposed to the incidents and accidents of the rough and tumble of life. Yet through each cross-section of the whole system there is delivered the same volume of blood in the unit of time. It seems hardly credible, but is a fact. How comes it about? The vasomotor system of nerves appears at first sight to dissociate, i.e. individualize, the different vascular areas, so as to meet their local needs, severally; here, by a spasm or increase of tone, which opposes a higher resistance to the ingress of blood into the particular area; there, by a relaxation of the walls of the vessels, with corresponding fall in the resistance and free flow of blood through the part. How is uniformity of the current as a whole compatible with such diversity of happenings? The problem seems more confounding than Samson's riddle.

## SIGNIFICANCE OF THE ANASTOMOTIC VESSELS

The anatomist comes to our aid; he shows us the beautiful contrivance of the anastomotic vessels, present in greater or less degree in every part of the body, which, by means of its auxiliary channels, places its large reserves of compensatory adjustment at the disposal of the circulation as a whole. The vasomotor nerves acting through these show us now their powers of co-ordination of part with part, and accordingly we witness how the blood, finding itself unduly opposed in a given area, seeks and finds an outlet through adjacent channels relatively more open:

this greater freedom of passage may or may not need the active intervention of the vasomotor nerves. In like manner, the resistance in a given area falling, the increased local flow, which must follow, will automatically deplete the adjacent vascular areas. It will be obvious how in this way the uniform delivery of the blood will be favoured, this being a mechanical need essential to the regular and equal contractions of the ventricles and the maintenance of a stable blood pressure.

## UTILIZATION OF THE BLOOD AS A CARRIER OF REMEDIES

This is perhaps an opportune moment to digress from the main argument and consider the practical application of the time relations of the blood circuit in our use of the blood stream as a carrier of remedies. Reverting to diagram 19 on p. 93, it may be said that we never attempt the introduction of the medicament between A and B, i.e. into the arteries of the systemic circuit. The reason for this avoidance is, in part, that these are ill adapted for the purpose by reason of the high blood pressure within them, and the consequent difficulty of controlling the escape of blood; in part, that the thicker walls, the average smaller calibre, and the deeper placing do not favour their selection; but a more valid reason is that the remedy so introduced would be liable to be held up in individual capillary areas (especially if there happened to be a marked affinity between the remedy and the tissue cells of such areas), and that in this way the general distribution of the medicament would be hindered.

Neither is the capillary area of the systemic circuit selected as a rule, though it is quite common to inject deeply into the muscular tissue. When, however, this method is adopted, a varying proportion of the injection may find its way into the capillary network, but the probability is that the chief part will enter the circulation via the lymphatic lacunæ (as in the hypodermic method) and a much smaller part by the venules.

The commonest modes of introduction are into the subcutaneous tissue or direct into the veins. The former is dealt with in the main by the lymphatic system, and, owing to the sluggishness of the lymph stream and the circuitousness of the route, it is relatively slow. It may be appreciably quickened by massaging the site of the injection, and so spreading the surface of absorption; in the end, after many or fewer halts by the way at interposed lymphatic glands, the remedy enters the blood current at the great veins at the root of the neck. Lymphatic absorption is at best a roundabout route, but for most purposes it is sufficiently rapid, and very appreciably quicker than absorption through skin or mucous membrane; it has, moreover, the great advantage that, the medicament being introduced *sub cute* into an area of minimal metabolic activities, is much less exposed to changes which would be likely to react unfavourably upon its active principles, than it would be within the alimentary tract or upon the surface of the body; it thus enters the circulation comparatively unaltered.

There remains the intravenous injection, i.e.

the introduction between c and d, on the right heart side of the capillaries (see Fig. 19). Obviously the nearer we get to the right heart, as in segments 23 and 24, the more rapid the effect, and whenever we are up against a crisis, as in acute heart failure, this is the route to be selected. As to choice of site, there is little time-advantage between injection into the veins at the root of the neck and those at the bend of the elbow, whereas the latter method presents many advantages in that the veins at the elbow are much more accessible and under control, and that the danger of the entry of air, which the negative intra-thoracic pressure imperils (especially during violent respiratory movements), is practically abolished.

What is the course of the remedy by this method ? It will be a very rapid one to the right heart, but it will still have to pass through the whole of the pulmonary circuit before it can be distributed to the body at large. The circuit, however, is a relatively short one, and we may note that the capillaries through which the medicament will pass will be those which are spread out over the air cells, and will not include those which derive from the bronchial arteries, which feed the substance proper of the lung framework ; true, there may be some inosculations of the two sets of capillaries where they adjoin near the air cells. This route will not subject the remedy to any disturbing activities excepting such as belong to the respiratory process, the dominant factor in which appears to be oxygenation.

The time occupied by this shortest route, *according to the schema*, will be about ten to twelve seconds before the remedy enters the left ventricle.

This time will, of course, depend upon the proportion borne by the volume of blood contained in the pulmonary circuit to the blood-volume in the systemic circuit, and, as already explained, the proportion between these two in the schema is arbitrarily assumed. The left ventricle will now deliver its blood (containing the remedy) into the root of the aorta, where, immediately, it will find access to the coronaries, and, in the share of blood apportioned to these, will enter upon its passage through the heart. There will be the usual delay before the cardiac capillaries are reached, but it should be noted that the capillaries of the heart will be the first of the capillary areas to feel the incidence of the medicament, the precedence increasing as the mouths of the supply-arteries to the different organs are more distant from the points of origin of the coronary arteries.

How soon will be the systemic response to the stimulus conveyed, whether excitant or depressant? Practical experience must measure this, and we shall be prepared to find a wide variability, according to the nature of the stimulus and the excitability of the tissue affected; and according, also, as the effect sought is direct upon the part as the drug circulates through its tissues, or indirect, i.e. the result of action conveyed from a distance through nerve centre and nerve tract: or whether, again, both modes of action come into play. Practical medicine and experimental pharmacology must speak here; but in respect of one organ at least, viz. the heart, we shall always select the intravenous method, when up against a crisis, and we shall do this, not merely

because it is the shortest route to the heart capillaries, but because we shall hope that as the remedy circulates through the chambers of the heart it may directly influence the walls across the endocardium, thus anticipating the capillary action within the heart substance. For this hope there is demonstrable evidence in the case of the isolated frog-heart through which an artificial blood is circulated by means of the Roy-apparatus. In this experiment the excised heart is tied on to a perfusion cannula and then placed in an oil chamber with movable bottom, to which a light lever is attached. Through the perfusion cannula the artificial blood enters and leaves the heart. Thus circumstanced, the heart beats freely, and its pulsations are recorded by a lever upon a revolving drum. The experiment consisted in throwing into the afferent blood stream near the heart a given quantity of an ammonium salt (stimulant) or of a potassium salt (depressant) and carefully noting the result.[1] There followed immediately, at an interval of time corresponding to the length of afferent tube to be compassed, a strengthening or weakening of the beat due unmistakably to the access of the drug to the cavity of the ventricle. As upon this the stream from the uncontaminated reservoir of blood washed through the ventricle, so the effect disappeared, the beat returning to the normal. Again and again this experiment could be repeated with identical

[1] This experiment was performed by the writer when working with Dr. Sydney Ringer, in the Physiological Laboratory of University College, upon the effects of various drugs upon the frog-heart.

result; and here, clearly, there could be no question of any capillary circulation through the walls of this excised heart. The effect could only be direct and from within the heart chamber. It will be said, there is a great difference between the delicate sponge-work of the frog's ventricle and the walls of the mammalian heart with their protective endocardial covering, but for all that we cannot reasonably exclude a direct action across the endocardium, especially when dealing with an appreciable quantity of a powerful stimulant such as ammonia,[1] thrown directly into a vein near the heart; remembering, as we must, that the given dose will remain relatively concentrated within the limited volume of blood which immediately receives it.

Let us follow more precisely the course of the medicament thus introduced. It has been said that there will be an interval of some ten to twelve seconds[2] before the left ventricle is reached —that is so; but within the pericardium there are two distinct hearts, and the first of these to feel the incidence of the stimulus will be the right ventricle. This will be reached at a much shorter

[1] Halford's recommendation, in the extreme circulatory collapse of snake-poisoning, was to inject 12 minims of the Liquor Ammoniæ Fortior of the B.P., diluted with three times its volume of water, directly into a vein. This would be the equivalent of about 40 minims of the ordinary Liquor Ammoniæ B.P. undiluted and 160 minims of sal volatile (Spiritus Ammoniæ Aromaticus B.P.) so far as the ammonia content goes. So pungent a dose could hardly fail to make itself felt within the heart, though diluted by 4 ounces of blood, i.e. the contents of auricle or ventricle.

[2] These figures refer to the normal state, but in heart failure the circulation is certain to be more sluggish, to what degree it is impossible to state.

interval, say two to four seconds (cf. the schema), and in this short interval the concentration of the drug will have suffered very little loss, diffusion being the chief means of dilution. We may take it, then, that as the medicated blood fills, first the right auricle and then the right ventricle, it will find its chief opportunity for direct stimulation of the walls of the right heart. This stimulation will be the more needed because in nearly every form of acute circulatory failure the blood tends to lag and gather on the right side of the heart, overloading its cavities; this, then, will be a great gain. But what about the left ventricle: must it wait for any similar action upon its walls, until the same quantum of blood has been passed through the pulmonary circuit, where in its passage through the close-set meshwork of capillaries it will have been exposed to very appreciable loss of its stimulant powers by evaporation (this will hold specially when the medicament is a diffusible stimulant, such as ammonia or ether)? This raises a very interesting and important point, viz. the possibility that direct stimulation of the right side of the heart may fall simultaneously upon both hearts. This possibility becomes, indeed, a matter of great likelihood when we consider that the sino-auricular node, the auriculo-ventricular node and bundle are situate all on the right side of the heart beneath the endocardium, and that the *a–v* bundle supplies both ventricles with a rich plexus of ramifications; it would therefore be surprising indeed if this sensitive tissue should escape incitation by the stimulus contained in the blood, and granting the incidence of the

stimulus upon the bundle, the effect upon both ventricles would follow of necessity. The conclusion, then, is that by the injection of a stimulant into a systemic vein, we may reasonably expect the speediest action upon the heart, and that this will include simultaneous action, via the *a–v* bundle, upon both ventricles. Such stimulation should be reserved for the acutest forms of heart failure.

After this action the medicated blood has still to traverse the pulmonary circuit before it is delivered into the left ventricle. Here, despite loss by evaporation across the capillaries of the lungs, there may still remain sufficient even of a volatile reagent to act directly on the walls of the heart, augmenting the preceding effect. The final or third opportunity for local action will arrive when the medicated blood is distributed through the capillaries of the heart; and let us note again that this action will be *simultaneous* upon both ventricles.

No mention has been made thus far of the introduction of the medicament directly into the pulmonary capillaries. This can be done, but only when the reagent is in the state of vapour, and though this method seems to afford a very direct route to the left ventricle (cf. schema), it will not compete with the intravenous method, (1) because the blood in the capillaries is moving at its slowest; (2) because the tenuity of the vaporous state handicaps severely the rate of introduction quantitatively, as compared with the intravenous injection of the active substance in liquid form. The method by inhalation has its

special indications as exemplified in the administration of anæsthetics.

In collapse the dynamics of the heart are threatened with extinction; the condition is one of adynamia, and the problem, the reinforcement of the waning powers—to that end no means may be neglected. We have just discussed how the blood stream may be utilized to bring to bear more or less rapidly certain cardiac influences, but in order to amplify the subject of cardiac stimulation we may anticipate the consideration of other cardiac aspects, to be treated later, by the briefest reference to reflex action as an energizing means. The theory of stimulation rests upon the assumption of reserve powers in the tissues to be stimulated; if there are none, the stimulant is valueless. The stimulant may act directly or indirectly, *after absorption* into the system; this has been dealt with. But it may act independently of any absorption, at arm's length, so to speak, as, for instance, when a strong solution of ammonia is introduced into the nose on a feather. In this case the violent local irritation is projected upon the cerebrospinal centres and thence reflected upon the heart (or lungs as the case may be) as a stimulant force. The phial of smelling salts was at one time much in vogue for attacks of faintness: it remains an adjuvant restorative of value—at times of the greatest value.

### OBSTRUCTION IN ARTERIES AND VEINS

This completes the digression, though perhaps it hardly deserves this name, being illustrative rather of the circulatory act.

Let us now revert to the main subject, the circulation through the vessels, and regard certain morbid happenings and the circulatory response. We have seen the value of the anastomotic system of vessels as ensuring the delivery into the heart of a uniform volume of blood, in the unit of time and across the vascular circuit, viewed as a whole; and this, despite incidental impediments to the flow in this or that locality. We are now to see another function which the anastomoses may perform, and how they may save the life of a blocked area, or, failing this, may reduce the area of destruction. The former of these acts concerns the life of the body as a whole, the latter is essentially a local act. The arteries come first for consideration.

The arterial anastomoses tend to increase as the capillary network is neared, being most numerous between the arterioles. By means of these pre-capillary communicating branchings and, in the last instance, by means of the capillary inosculations with neighbouring capillary areas, all arteries do intercommunicate. Whence it comes that a very thin fluid injected will tend to find its way from any given part into every other part of the body. In addition to these finer intercommunications there are anastomoses between neighbouring arteries of larger calibre. They are of two kinds: inosculations of single branchings, generally of appreciable size, in the form of arches. From these arches offshoots may be given off, which, after bifurcating, may repeat the process of single inosculation with adjacent arteries of their own order of magnitude. The other form of anastomosis, between vessels

of size, may be by a network of branching communications, which in type approach the pre-capillary networks.[1]

In contrast with these modes of connecting up adjacent or even distant vascular areas, which constitute the rule, there are found, exceptionally, capillary areas which are relatively shut off from their neighbours in so far as their supplying arteries are concerned; such arteries are termed end arteries.

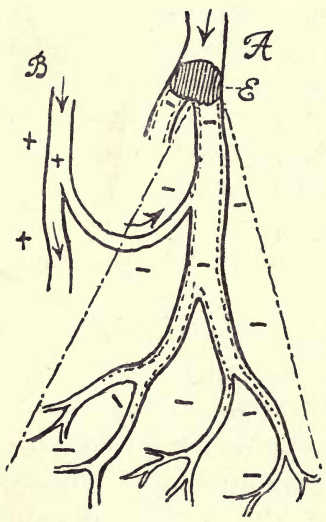


FIG. 20.—Schematic representation of the effects of arterial blocking of an artery, A.

In the following diagram one type of arterial anastomosis has been chosen to stand for all kinds, viz. that by the inosculation of single vessels; since on the question of intercommunication we are concerned only with the freedom or restriction of the communication, which alone governs the effects, the form of communication is immaterial to the argument.

Anastomosis with a neighbouring artery, B, is shown in the form of a direct arch-like inosculation. The embolus, E, is seen *in situ*, lodged at a bifurcation. The diagram presents the *immediate* effects of the blocking; thus within the vessels, below the block, the minus sign indicates the lowered blood pressure which exists,

[1] Henle, op. cit., pp. 68, 69.

and the same sign outside the vessels signifies that in the whole of the vascular area supplied by the occluded artery, the same minus pressure obtains (this area is represented as triangular in shape). The dotted lines within the vessels are intended to show the contraction of the arteries. Outside the blocked area the plus sign shows the relatively raised blood pressure present there, and the direction of the arrow shows the flow of blood caused by this raised pressure into the *locus minoris resistentiæ* of the branchings of A below the block.

Such are the immediate effects, for it is clear that, the arteries being under a distending pressure up to the moment of the occlusion, the sudden withdrawal of this pressure must be followed by the recoil of the stretched walls, and this effect (shrinkage) will move, *pari passu*, as the blood pressure continues to fall within the ramifications of A.

Now let us suppose that the collateral anastomoses of A are sufficiently free to remedy this state of things; in that case the circulatory embarrassment is just a temporary matter which soon passes, the blood pressure differences between the areas of A and B being soon equalized, and thus the effects of the embolism nullified. But supposing them to be insufficient for this purpose, then a condition of relative stagnation sets in within the branchings of A, in consequence of which the tissues, including the walls of the vessels, suffer in their vitality and the whole area becomes toneless. Hence, though the anastomoses are inadequate to effect a good circulation, the slack vessels will yield to the pressure of

invading small streams of blood, with the result that the part becomes overcharged with blood moving more or less sluggishly or completely arrested. This penetration from contiguous areas must continue so long as there is vascular continuity between the part and its surroundings, so long as the blood is liquid, and so long as the intravascular pressures are unequalized. It is the end arteries which furnish the most extreme forms of ischæmia as the result of blocking, and it is in them that we meet with the infarct, so called, in its most pronounced form. Here, in the absence of any sufficient collateral anastomoses, the filling and overfilling of the blocked area can only take place by regurgitation through the capillaries: the inexorable laws of physics will compel this.

We pass to the veins. These vessels are amply supplied with anastomoses, which carry out the same purpose as the arterial anastomoses in respect of the circulation of the blood as a whole; indeed, unless this were so, the arterial safeguards would be quite fruitless. We may therefore pass at once to the mode of working of the venous intercommunications as they affect the flow through a blocked area.

The following is the sequence of events as observed after the ligature of a vein (the obstruction by a thrombus would give similar results, though less strikingly, because they would develop less suddenly).

The movement of the blood is stayed behind the obstruction, and in consequence the *vis a tergo*, which until that moment had been expending

itself in developing a liquid momentum (the blood current), and also in overcoming the surface resistance of the walls of the veins to this current, now spends itself wholly upon the walls of the vein as a distending force, to which they yield: the vein therefore swells immediately behind the obstruction. This process is repeated backwards, towards the capillaries, as in turn each fresh influx of blood from the capillaries comes up against a wall of stationary blood. The distension of the veins therefore spreads peripherally until the capillaries are reached; these now become distended in their turn, for the blood pressure in the arteries supplying the area in question finds no other outlet than as a distending force.[1] We have now passed from a state of liquid movement to one of stasis more or less complete, from a problem in dynamics to one in statics. Meanwhile the *vis a tergo* has remained unchanged, i.e. the force of the blood pressure in the arteries which plays upon the blocked area, and the change amounts simply to this, that all current having been abolished in this area and all friction, depending upon current, the unspent *vis a tergo* manifests itself in a gradual *rise of blood pressure* in the whole of the vascular area which the blocked vein has ceased to drain.

The picture presented to us shows a canalized area, overcharged with stagnant blood, the result of blocking of the main drain. This area

[1] The actual distension of the veins will increase as the stasis persists, owing to a loss of tone in the walls of the veins, the direct result of the stagnation which occasions a lowering in all the vital activities of the part.

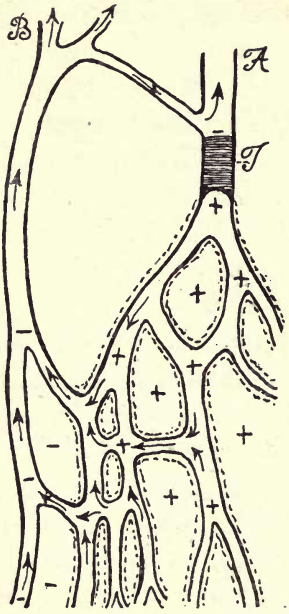


FIG. 21.—Semi-diagrammatic. Two neighbouring veins, A and B, are shown with anastomoses at three places. A is blocked by a thrombus at T. Below the block there is stasis in all the branchings of A, and the swelling of these veins, under the tension marked by the signs + +, present in the whole of the area of stasis, is indicated by the dotted lines. The + signs in the meshes of the venous branchings indicate the same rise of pressure in the venules (not shown) and in the extravascular spaces. The — signs within B and its branchings, as also within A, just above the thrombus, indicate the minus pressure there prevailing. The arrows indicate the lines of flow, or rather the lines of pressure, for the pressure may not take effect. Contrasting this figure with that of the diagram showing the effects of arterial blocking, we observe that the two are direct opposites.

is surrounded on all sides by similar canalized areas with which it is in communication, more or less free, by means of subsidiary channels. In the whole of the stagnant area the fluid pressure within the channels is raised above that of the surrounding areas, through the channels of which an uninterrupted flow is maintained. Thus stated we behold a condition of unstable equilibrium which must tend to right itself. Let us consider how this comes to pass, and how the *vis medicatrix naturæ* asserts itself: a diagram will assist.

It has been said that the blocking of the vein A develops a state of unstable equilibrium between the area drained by it and the adjacent areas drained by veins with which it is in direct communication; the unstable

equilibrium results from the rise in blood pressure occasioned by the block; this rise in blood pressure figures as the *genius loci*, alias the *vis medicatrix naturæ*. Let us note its mode of operating.

Now the very first effect of the blood pressure rise will develop on the distal side of the block, because rise in pressure begins here. This effect will go on, continuously increasing, until the maximum rise has obtained, and throughout the whole period its tendency will be to shift the block. It must be noted here that on the proximal side of the block immediately above it, the fall in blood pressure begins simultaneously with the rise just below the block, and that this fall will continue until the anastomoses have established an equilibrium.

We must next note that in this tendency to shift the obstruction, the *genius loci* is very parochial in its outlook, taking cognizance of the advantage of the part alone, and thereby forfeiting its right to the title *vis medicatrix*. For should its efforts succeed in dislodging the obstruction, whilst bringing immediate relief to the part, as it would truly, it would at the same time imperil other parts, probably more vital, by chasing the thrombus into the circulation and converting it into an embolus; indeed, if the block were of sufficient magnitude it would directly endanger life. Our efforts, therefore, must always be to thwart this solution of the problem.

Failing this mode of action, we find ourselves on safer ground as we witness the operation of the raised blood pressure taking effect at the periphery of the blocked area and forcing its way

along the lines of least resistance, i.e. the anastomotic communications. In this way the congested part unloads itself safely and more or less effectively, according to the freedom of these channels. We need not pursue this operation except to point out that as it takes place the neighbouring veins which do the unloading become themselves temporarily congested and raised as to their blood pressure, and to draw attention to the working of the upper anastomosis in relieving this temporary congestion by utilizing the main trunk of A just above the thrombus as a draw-off. In these acts Nature re-establishes her claim to be a true *vis medicatrix*.

The blood pressure rise in the area of congestion is by no means a negligible quantity; thus in the capillary area corresponding to the blocked vein it may amount to a quadrupling of the normal pressure [1]; and we may note that the greater the stasis, i.e. the fewer the adjuvant anastomoses, the more will the capillary pressure be approximated in the derivative veins, since in fluids at rest the pressure is uniformly distributed.

In the extremest forms of congestion through deficiency of collateral supply channels, whether in the arterial or venous system, the death of the part must ensue.

As we proceed along the veins toward the heart, and the channels become wider and wider, so the need of the anastomotic relief vessels becomes more and more essential in direct proportion to the magnitude of the ischæmic area, and hence we find them more and more in evidence even up to the atrium of the heart,

[1] Halliburton, *Physiology*, ed. 1917, p. 278.

and the blocking of one or other vena cava. For not only must the blood be kept on the move through the tissues for the sake of their vitality, but the delivery into the heart must also be kept up to a certain standard or the ventricles would not have the wherewithal to contract upon. In the extremest forms of blocking we find the blood taking the most circuitous routes; as when the superior cava unloads its surcharged area into the tributaries of the inferior cava, or *vice versâ*. Have we perhaps here in this safeguard the reason for the separation of the two cavæ instead of their union in a common trunk?

Different levels of blood-pressure and lines of greater and less resistance, these are the sole determinants of the flow measured by volume and by current rate, as we witness it from aorta to capillary and from capillary to the mouths of the cavæ.

## OBSTRUCTION IN THE HEART CHAMBERS AND BACK-PRESSURE EFFECTS

And now, leaving the vessels and entering the chambers of the heart, let us consider the effects of impediments placed here or there.

Let us suppose an obstruction on the arterial side at the root of the aorta, what will ensue? Death, if the blocking be complete, for no by-way presents, anastomosis cannot come to our aid; the circulation is arrested at its source. But, short of such complete occlusion, let us take a case of narrowing of the aortic orifice by thickening and fusion of the segments of its valve, and assume that the condition will have arisen and progressed slowly; what will be the result? In

this case a resistance will have been thrown into the circuit which has had to be met. Again, we note that anastomosis can afford no relief. Now if, previous to the arising of the obstruction, the heart's contractions were sufficient to yield an adequate circulation, they must fail now, for the load has been added to, consequently the circulation must there and then, in greater or less degree, become ineffectual, and this must show itself in the usual way. But this is not what we do find in fact; on the contrary, the circulation may be perfect, however severely it may have been tested. The explanation discloses itself in the discovery of another compensatory mechanism in the shape of the potency of the heart muscle to grow, it matters not how, in exact proportion to the rise in the resistance which it is called upon to overcome—within limits, of course, for here is no cornucopia. Thus stated we have the law of compensatory hypertrophy, which applies to all hollow muscular viscera. This reserve power may suffice for a whole lifetime with little appreciable discomfort or restraint; but in general, in the course of time, it calls for restriction of the demands made upon the heart in order to eke out existence upon a store, which, as stated, is limited. In other cases, more severe in type, even the strictest economy may not suffice, and then slowly but surely the heart fails to meet the situation, and its compensation breaks down.[1]

The left ventricle will by then have become

[1] More often, probably, in these cases of aortic stenosis the disease is latent for many years, and the failure is apt to manifest itself comparatively acutely (*vide supra*, Dr. Peacock's cases).

unable to empty itself sufficiently,[1] and the blood will tend to accumulate within, distending the walls of its cavity. The left auricle will next follow suit, and the distension of its cavity will hold up, more or less, the blood of the pulmonary circuit. As this holding up is propagated backwards, we are brought to the right ventricle, which now in its turn becomes over-distended, and thereupon the right auricle likewise. The venæ cavæ become next affected, simultaneously, and we witness in them and their tributaries all the effects which follow upon the ligation or thrombosis of a peripheral vein, with this exception, however, that, since the whole systemic venous system is equally involved, anastomosis can avail nothing, and the effects are felt right down to remotest venules with their capillary areas. Briefly stated, these effects are, stases, hyperæmias, cyanoses, œdemas, added to which is the tendency of the congested areas to develop low forms of inflammation.

It is of interest to note that, though the entire venous system feels the effects of the obstruction at the heart, certain areas tend to exhibit them more strikingly than others, i.e. there are sites of election. Thus in the pulmonary circuit it is the bases of the lungs posteriorly which are particularly selected, whilst in the systemic circuit it is the lower extremities and the more dependent parts of the body in general, or else the portal area, which are chosen. Why this happens in a given case may at times be wholly inexplicable,

[1] It is necessary to put it thus, for it is not known whether a perfectly normal heart, which has made no call on its reserves, does empty itself completely at each systole.

and we are then driven to postulate in the area in question a lessened power of resistance in venules and capillaries, which belongs to the original make-up of the body. In other cases the local œdema or congestion is more obviously explained by the presence of adverse conditions such as dependence and immobility, as in the case of the bases of the lungs posteriorly, with dorsal decubitus; conditions which clearly handicap the part.

Now in these manifestations have we evidence, or have we not, of a circulatory failure? And these same manifestations, have they, or have they not, been propagated backwards from the site of the obstruction? And are they, or are they not, the result of a blood pressure rise on the venous side of the circuit? If the answer to these questions is in the affirmative, then could we designate them better than as "back-pressure effects"? And since the heart is the organ which is responsible for the proper circulation of the blood, why not say that, all compensatory mechanisms having broken down, we are face to face with heart failure; and that according to their degree these manifestations measure the extent of the failure?

It is necessary to dwell on this subject because "the theory of back pressure" has been impugned of late and made responsible for serious misconceptions, so much so that it has been questioned whether the discovery of auscultation (upon which we rely so much for detecting local obstructions and incompetencies in the heart) "has done more harm than good" to the advancement of the science of medicine. And do we not

actually introduce a misconception when we speak of the "theory" of back pressure, for when we describe the above-named manifestations, we advance no theory; we simply say, here *is* circulatory failure, and that *ipso facto* and *pro tanto* it shows heart failure. For just as, when we see a flying ball rebound from a surface which opposes its course, we say that the rebound declares the resistance the ball has met with; so when we witness the recoil of a venous ischæmia from the heart, against stream, and discover by physical signs the evidence of an impediment at the cardiac valves, whether due to obstruction or incompetence it matters not, we say that it is the interposed resistance which is causal. If it be argued that all these effects may arise without any mechanical obstruction *within* the heart, as in the case of a thickened adherent pericardium, we shall of course agree, but in that case the crippling of the fibres of the heart from *without* opposes their effective contraction as markedly and as mechanically as the internal resistance at the valve. Again, if it be urged that the same effects may be witnessed in the absence of any mechanical impediment, whether within or without, we shall again agree, and shall be compelled to infer a primary failure of the heart muscle, toxic or degenerate, as the case may be; and once more this in no wise vitiates the argument which other cases show of mechanical obstruction as a primary event in the chain of cause and effect. For after all we are faced ultimately, in every case, with the failure of the muscular fibre; this must be so, seeing that the circulation is the outcome of a

muscular act, and only through default or insufficiency of that act can circulatory failure arise. The crux, then, of the whole matter lies just here, that, looking at the cases of circulatory failure as they appear before us clinically, we see in certain of them a mechanical resistance at a valve; in certain others a mechanical impediment from without; in a third variety a failure of the muscular fibre itself—and one or other of these we see as the *primary* causal event in the morbid sequence.[1]

A caveat must be entered here; it is not contended that every case of heart failure must show back-pressure signs, for we know that these signs are in general slow in their inception and progress, and there may not be the necessary time to develop them. Thus in collapse from shock, e.g. from snake poison or other intense toxæmia, there may be the profoundest heart failure without any sign of back pressure: these cases are rapid in their course whether towards recovery or death. But the time element is not the only reason for the absence here of the back-pressure effects, for if we look at the conditions which obtain, we see that they contrast most strikingly with those which are present in the typical case of valve heart failure. Thus, in the first place there is nowhere any mechanical obstruction to raise a barrier and generate a

[1] We are familiar with the same kind of thing in the case of other hollow muscular viscera, as, for instance, when there is bowel failure due to mechanical obstruction which the peristalsis of healthy fibres, however forcible, is unable to overcome, as in strangulated hernia; and, on the other hand, when the bowel is unable to pass on its contents because of a paresis or paralysis of the muscle fibres of the gut, there being no mechanical block.

local plus pressure behind it; and, next, there is an enormous blood-pressure fall due to a vasomotor paralysis affecting both arteries and veins; and though the blood tends still to accumulate on the venous side, in particular in the capacious abdominal veins, these are so slack and so capacious that there is not blood enough to distend them under pressure. Such cases have been described as "bleeding into their own veins." In the absence, then, of raised venous pressure, how should we look for back-pressure effects?

It may be noted that under such conditions the supply of blood to the heart itself will be insufficient, and that the ill-filled organ will be unable to beat up a sufficient intracardiac pressure, thus completing the vicious circle. Escape from this circle is only possible on the terms (1) of a vasomotor stimulation which shall cause the arteries and veins to contract, narrowing their bed, as by the hypodermic use of pituitrin and physostigmine and remedies of their class; and (2) by the stimulation of the heart muscle to more vigorous action, as by strychnine, caffeine, or camphor.

There remains one other aspect of this subject which calls for consideration and comment. It has been objected that, "even in cases where the exhaustion may be so extreme that death impends from heart failure, the heart is spoken of as having good compensation because none of the 'back-pressure' symptoms are present."[1] And again, on p. 7 of the same work we find

[1] Sir James Mackenzie, *Diseases of the Heart*, 1913, p. 6.

the statement: " . . . while in the great number of cases of grave heart failure so-called signs of back pressure are entirely absent." Now to what class of case do these statements refer? Do they include the acute fulminating forms of heart failure just described, those accompanied by symptoms of profound collapse and running a rapid course, or is the reference to cases of more protracted course? I suspect the latter because, in the same sentence from which the last statement has been taken, the words occur, " when I have watched the progress of heart failure," and I cannot help thinking that there is some confusion here, and that what is really in mind is heart disease, and not heart failure. For that heart disease may be present in gravest form up to the verge of impending death, without any symptoms of back pressure, none will deny who are familiar with aortic regurgitation as a cause of sudden death. Such cases are frequent though symptoms are completely absent, even when examination (as at Insurance Offices) may discover not only pronounced signs of the disease, but as pronounced signs of the heart's reaction to the strain put upon it, in the shape of dilatation and hypertrophy. The type of the pulse in such cases may be very characteristic, if not pathognomonic, of the disease, but so long as the rhythm is regular and the lift forcible (though not sustained) and an average blood pressure is maintained, sufficient to give a good flow of blood through the tissues without any lagging on the venous side; and, most crucial of all, so long as the general metabolism is at normal and no subjective disabilities are

present—where is the heart failure? A type of pulse may point to disease and warn us of dangers ahead, possible or probable, in the same way that "the sudden inception of a new rhythm"[1] may give like indications; but we are still not in the presence of heart failure unless it can be shown that there is actual failure to maintain an efficient circulation. The problem is a purely mechanical one: either the heart develops sufficient energy to overcome the inertia of the blood and the resistance of the tubes, or it does not.

It has been said that in every case of circulatory failure we are ultimately faced by failure of the heart fibre. This failure may be primary at the heart fibre, or it may be there secondarily in sequence to causes elsewhere. In the latter class we place the cases of circulatory failure which arise in the course of valvular disease of the heart. Such disease throws into the circuit an added resistance which must be met by the heart fibre; is it unreasonable to suggest that the continued effort which a permanent resistance demands may in the end lead up to exhaustion of the fibre with accompanying or preceding degenerative molecular changes?

[1] Mackenzie, op. cit., p. 7.

## CHAPTER IV

### UPON CERTAIN POINTS IN CONNEXION WITH VENOUS PULSATIONS AND VENOUS TRACINGS

A good many years ago Dr. Sydney Ringer, in conjunction with the writer, contributed a paper [1] upon "The Pulsations and Murmurs in the Great Veins," in which our consideration was confined practically to the veins at the root of the neck. Since then much work has been bestowed upon the subject, which, indeed, has assumed a very prominent position. I am anxious now, having regard to this prominence, to bring certain aspects of the question before my colleagues, and to this end would propose that we follow the course of the blood in the veins, from its origin in the capillaries until it is about to enter the thoracic cavity.

Excluding rare and abnormal states, the venous flow, as it issues from the capillaries, presents a uniform stream which gradually gathers pace as by confluence the venules become veins, and these in turn, by a progressive union of neighbouring channels, form larger and larger trunks. The gathering of pace by the current corresponds with the narrowing of the bed of the stream. The set of the flow is always towards the intrathoracic cavity, and the determinant of this course is the low pressure which prevails in that

[1] *Trans. Med. Chir. Soc.*, 1892.

cavity, a pressure which is, in fact, negative to that of the atmosphere.[1] The determinant, however, is not a fixed quantity, increasing as it does when the cavity expands during inspiration, and diminishing as the chest walls subside in expiration. The effect of these variations is upon the whole venous current, but at its origin in the capillaries it would require the microscope to show it. In its nature it is neither a pulsation nor a thrill, but rather a waxing and a waning of the stream which resembles the flow and ebb of the tide, the one act merging insensibly into the other; it might be termed the respiratory venous tide. As the great veins near the thoracic cavity, the inspiratory acceleration of the blood may become audible as an increased loudness of the venous hum.

Thus far, until we arrive at the lower end of the neck just above the clavicle, the systolic drive of the left ventricle has brought the venous blood in a steady stream, in which no variation is observable save that of the gentle rise and fall of the respiratory tide. But now we approach the heart and come into touch with great and abrupt activities within its cavities, and inasmuch as a continuous stream of blood connects those cavities with the blood of their tributary veins, we must be prepared to find the cardiac activities reflected back a longer or shorter way into the veins, in the shape of pulsations or ripples, i.e. oscillatory movements coarser or finer, or even at times movements of actual translation, i.e. of reflux.

The abrupt intracardiac activities just men-

[1] At any rate during the inspiratory phase.

tioned are represented by the systoles of the right auricle and right ventricle; there are none other which can be propagated from within the heart along the channels of the veins to the root of the neck, and the pulsations to which they give rise might be called intrinsic, to distinguish them from those pulsations which the same veins may exhibit as the result of impulses which reach them from without, these pulsations being termed extrinsic. As examples of the latter we have the pulsations produced in the veins at the root of the neck by the throbbing of the contiguous arteries—carotid, subclavian, and innominate; these arterial pulsations derive from the left ventricle. Thus within a small area, which a two-shilling piece would cover, just above the inner end of the clavicle, we have the meeting-point of impulses from both sides of the heart, which play upon a thin-walled vessel, the internal jugular, more or less filled under low pressure. The conditions favour the production of oscillations within the vein, of a coarse undulating character, and these have scarce time to subside before the intracardiac activities of a fresh cardiac cycle renew the disturbances. It must be pointed out that the movements now under consideration are quite distinct from those slow movements of rise and fall which correspond to the swell and subsidence of the auricle, as its walls follow *passively* the venous inflow which the intravenous pressure determines; and quite distinct also from the variations in this venous inflow as it fluctuates with the respiratory phases: neither of these movements could beget the pulsations and oscillations in question.

*The auricular systole* falls in the late period of the ventricular diastole, and it is completed just before the ventricle begins to contract. In the normal condition of the heart, it is an act of small power, for it is only opposed by the slack walls of the ventricle and the almost negligible resistance of the wide auriculo-ventricular opening. Rarely is the tricuspid orifice sufficiently narrowed by disease to call forth an appreciable effort from the right auricle and lead up to a compensatory hypertrophy of its walls; it is otherwise with the mitral orifice, which is subject to disease and may thereby be constricted, not infrequently to an extreme degree: in such case a marked hypertrophy of the auricle may obtain.

The purpose of the auricular stroke is to propel the blood forwards into the ventricle and complete its load, i.e. its distension. This being so, and in order to render the stroke more effective, it is important to secure a base from which the stroke can take effect. It is clear that one could not advantageously fire a gun with an open breech, for by as much as the charge took effect backwards the forward projection would suffer: the breech, therefore, must first be closed. It is exactly the same for the auricular contraction, the mouths of the tributary veins must first be closed or reduced to a minimum in order that the purpose of the stroke may be the better fulfilled. If we consult the figures and text in Henle and in Quain, particularly the former, we see how the openings of the cavæ and of the coronary sinus into the right auricle and those of the pulmonary veins into the left auricle are provided with annular muscular fibres, the contraction of which

must have a sphincter-like action. This points to an order in the times of contraction of the auricular fibres, for it will be quite clear that the sphincter-action of the veins must precede that of the general contraction of the walls of the auricle, else Nature would thwart her own purpose and contrivance, which is not her wont. In fact, we may take it that the part played by these annular fibres surrounding the mouths of the veins is exactly the same for the auricle as that played by the auriculo-ventricular valves in the case of the ventricles—namely, to furnish either act with a taking-off ground for the power it develops.

But if this be true for the *normal* working of the heart, how much of the auricular systolic impulse will be propagated back into the supplying veins? Probably very little, if any. It will be different in abnormal states, and in proportion as a particular case may show an overloaded, imperfectly-contracting auricle with attendant distended veins, we shall expect to see a backward propagation of the auricular systole, as a more or less well-defined wave. This wave will precede the onset of the ventricular systole.

*The ventricular systole* is prepared for by the auricular systole, and as follows. The completion of the ventricular load will be attended by more or less distension of the slack ventricle, i.e. more or less physical stretch of its walls, and the moment the auricle relaxes its grasp (its systole completed), that very moment there will come into action the elastic recoil of the stretched walls, which recoil will float into apposition the

flaps of the auriculo-ventricular valve. The breech of the ventricular power-chamber is now closed, and all is in readiness for the next event, which follows immediately, namely, the ventricular systole. This act is not a single event, but is composed of a sequence of events which runs through the system of ventricular fibres: the reasons for this conclusion are given elsewhere. In its onset the act is abrupt, and it gathers force rapidly, the intraventricular pressure rising simultaneously. The strain put upon the *a–v* valve is equally abrupt in its arising, and under its influence the whole valve is made to bulge suddenly towards the auricle as far as the counteracting musculi papillares and the attachments, the chordæ tendineæ, will permit.[1] As to these two events, the movement backwards of the flaps of the *a–v* valve at the *end* of the auricular systole, and the subsequent accentuation of this same movement with the *onset* of the ventricular contraction, it is probable that the former is too weak a movement to yield an appreciable pulsation, apart from the fact that the auricle is more or less emptied, at the time when this pulsation would take origin, and, further, that the set of the current of blood into the auricle would oppose its propagation backwards. In respect of the latter, though we are dealing with a movement of considerable energy and suddenness capable of overcoming the contrary set of the current, we must note that it also would be opposed by the relative emptiness of the auricle still present. It is doubtful, there-

[1] The time of this backward thrust of the valve will obviously be early systolic.

fore, whether even this latter pulsation could record itself in the tributary veins under conditions of health; in morbid states, on the other hand, marked by dilated heart cavities, never emptying themselves effectively, and by valves which do not hold, this same would probably yield a definite wave accentuated by more or less of reflux.[1] The time of this pulsation would be early ventricular systolic.

Reverting to the normal working of the heart, we have noted the rapid rise in the intraventricular pressure under the force of the systole; this culminates in the forcing of the semilunar valves, till then securely held by the intra-arterial pressures. With this forcing, a wave is thrown into the arterial systems which is generally spoken of as systolic and is so, but it will be noted that it does not begin with the onset of the ventricular systole; it will be noted also that it is a wave which is propagated forwards, not backwards, and that it could not appear in the veins at all unless by juxtaposition the arterial wave could impinge upon a neighbouring vein. This contiguity is present, we know, at the root of the neck, and the arterial pulse must therefore communicate itself to the blood in the internal jugular vein.

Let us pass on to consider the question of the pulsations occurring in the great veins, in closest

[1] The reflux *wave* would probably arise as a kind of percussion wave at the first beginning of the reflux, and fall short of the duration of the whole reflux, which should be coextensive with the ventricular systole.

touch with the heart, and ask ourselves what we can learn clinically therefrom. In the first place we must note that, in so far as the activities of the right heart are concerned, the only veins accessible to our methods of examination are those at the root of the neck, and in particular the right internal jugular. It is true we can observe and record a hepatic pulsation, but we cannot get near the inferior vena cava as it approaches the heart, nor at the trunk of the hepatic veins.

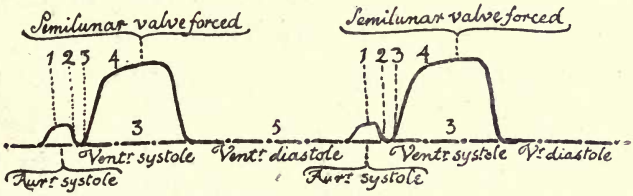


FIG. 22.—Schematic representation of the cardiac cycle. There is no attempt to represent the actual form of the curves of contraction, the purpose of the schema being simply to show the relative positions and durations of the auricular and ventricular systoles. The figures 1, 2, 3, 4, give the sequence of events capable of causing intravenous pulsations in the large veins near the heart. The position of 4, on the systolic curve of the ventricle, is quite arbitrary; it merely records the fact that the forcing of the semilunar valves is posterior to the onset of ventricular systole.

In the right internal jugular the following pulsations may theoretically appear; the numbers 1, 2, 3, 4, correspond to those in the schema:

### *Intrinsic Pulsations arising within the Cavities of the Right Side of the Heart*

1. A presystolic pulsation corresponding with the auricular systole; it is of small power normally.

2. A minute pulsation occasioned at the end of the auricular systole by the physical recoil of

the ventricle (still in relaxation and which the contraction of the auricle has put on the stretch). This recoil floats back the segments of the auriculo-ventricular valve into apposition.

3. A pulsation following immediately upon 2, caused by the onset of the ventricular systole, which accentuates the backward movement of the *a–v* valve initiated at the close of the auricular systole. This pulsation is of considerable force.

*Extrinsic Pulsation arising within the Left Ventricle*

4. A pulsation which arises within the left ventricle and is conveyed by the carotid and subclavian arteries to the root of the neck, and coming there into touch with the internal jugular vein. This pulsation is of considerable force and of higher tension than 3. It corresponds in time with the forcing of the aortic semilunar valves.

These pulsations, which may theoretically appear in the internal jugular, will be of very doubtful occurrence *in health*, when the arteries are well filled and the veins are relatively undistended. The likeliest to put in a demonstrable appearance will be No. 4, the extrinsic carotid pulsation. *In disease*, when the blood distribution tends to preponderate on the venous side, we should look for 1 and 3, especially the latter, the pulsation of which would be apt to be reinforced by more or less actual reflux. Pulsation No. 2 will probably be conspicuous by its absence. Pulsation No. 4 should be well marked.

Passing now to the left side of the heart, the same sequence of intrinsic venous pulsations must arise with the same theoretic backward projection. But how far this projection carries, whether in health or disease, it is impossible for us to demonstrate actually, seeing that these pulsations do not figure at all at the root of the neck, but are directed straight back into the pulmonary veins, and thence, via the roots of the lungs, into the depths of the thoracic cavity. Throughout the whole of this course the venous pulsations are inaccessible to our methods of examination, and we are therefore unable to inscribe them and bring them into juxtaposition with, say, the radial pulse tracing for purposes of comparison. Yet this is of the utmost importance, for what we call the heart is really two distinct hearts, each with its separate circuit. They are inseparably united, it is true, and their individual chambers—auricular, ventricular—work consentaneously; but they have different tasks and develop different quantities of energy, and in the development different rates of flow and different strengths of pulsation emerge—in all these directions the left heart greatly exceeding the right heart; moreover, in disease the morbid incidence is strikingly different, the left heart being much more prone to be affected. The individualization being so marked functionally, it is all the more important that we should be able to compare together the auriculo-ventricular events of each side of the heart separately. Thus in a case of mitral stenosis, which has given rise to a pronounced compensatory hypertrophy of the left auricle, the presystolic wave, which

must be greatly accentuated, is entirely concealed from us within the chest. Supposing now that in such a case a well-defined presystolic bruit have been present and should then suddenly disappear, we are quite unable to confirm the default of the left auricular systole, to which this lapse points by demonstrating the concomitant disappearance of the presystolic wave. In the very rare case of a tricuspid stenosis giving rise to a right auricular hypertrophy and a definite presystolic bruit, such as has been described by Dr. Gairdner, we should have this desired direct confirmation by the internal jugular vein at the root of the neck; but in the case of mitral stenosis we lack this direct evidence, and can only *surmise* that the disappearance of a much feebler presystolic pulsation in the neck, due to default of the right auricular systole, will have involved a like default of the left auricular systole. This is unsatisfactory; we want demonstration, not conjecture. In these remarks we are, of course, assuming that jugular tracings do show us unmistakably the auricular systolic wave.

The same will hold for the systolic venous pulse produced by regurgitation through the mitral valve; it will appear nowhere on the surface of the body, though it is quite likely that the lungs would show it, especially in their bases, in an even more striking way than does the tricuspid pulsation witnessed at the root of the neck, and occasionally in the hepatic area in cases of pronounced reflux.

This latency of the intrinsic venous pulsations of the left side of the heart holds so long as the

inter-auriculo-ventricular septum is intact.[1] It will be self-evident, however, that any malformation of the heart which destroys the integrity of this septum, as by a patent foramen ovale or by a defect in the interventricular septum, will put the left side of the heart in direct communication with the superior and inferior venæ cavæ, and so the intrinsic pulsations from both sides of the heart will figure together in the areas of distribution of these veins.

Thus far no mention has been made of fibrillation of the auricles as a cause of venous pulsations in the great veins. On the face of it, it is hard to comprehend how such mere flickerings of fibres could register their tremolo, seeing that they are minimal when compared with the intrinsic pulsations which we have been discussing, considering also that whilst the intrinsic pulsations are concerted acts the fibrillations are entirely incoherent, disconnected. Repeating what has been once before alluded to: if these flickerings resemble in any way the inco-ordinate contractings and relaxings of the walls of the ventricle of the frog-heart poisoned by digitalis, they must be mutually annulling. Yet we see these fibrillary waves figured in tracings obtained

[1] In conflict with this statement are the tracings given on p. 269, *Diseases of the Heart*, Sir James Mackenzie, 3rd ed., 1913. The upper tracing purports to show the effect of the auricular systoles (which have been blocked) upon the radial tracing. It is expressly stated here that the indentations on the falling line of the tracing are due to the left auricle. The second tracing, underneath, gives the radial tracing and that of the internal jugular from a similar case of heart block. It is confirmatory of the foregoing, but the auricular systoles are not qualified as being those of the *left* auricle.

from the root of the neck and even competing in size with the "*c*," "*v*," waves.[1]

RELATIVE VALUES OF THE VENOUS PULSATIONS

On this question of the magnitude of the individual waves of the venous tracing, and reverting to the intrinsic pulsations, it must be confessed that the results obtained are sufficiently perplexing, when we compare the relative values of the waves "*a*," "*v*," and "*c*," i.e. auricular systolic, ventricular systolic, and carotid. If we recall the type of heart from which these tracings are most commonly obtained, viz. the heart in distress; overloaded, in particular as to its right side; ineffective in the emptying of its cavities during the systoles, auricular and ventricular, and again in particular as to the right side;—if we consider next that cavities with thinner walls will tend to exhibit greater dilatation, and for this reason will be the less effective in passing on their contents, we shall surely conclude that the disproportion between the force of the auricular systole and that of the ventricular will, in such overloaded hearts, be markedly accentuated, i.e. the auricular systole will be by so much the more feeble, and of course the auricular wave likewise.

Passing to the wave "*v*," the ventricular wave, "so named because of its association with the systole of the right ventricle,"[2] we shall note first that it is caused by the forcible driving back

[1] Cf. Mackenzie, op. cit., p. 221; note in particular the middle tracing.

[2] Ibid., p. 150.

of the tricuspid valve, as the ventricle enters into systole; next that it begins, therefore, with the very first part of the ventricular systole; and, thirdly, that if the valve is not competent, more or less reflux will accompany the backward drive of the valve and reinforce its wave; fourthly, that the more or less persistent overloading of the right auricle and its tributary veins will facilitate the backward transmission of the wave so produced.[1] The ventricular wave, "*v*," should therefore always appear with the very first beginning of the ventricular systole, and if there is reflux, it also will begin at the same moment, but this reflux will prolong the wave, since it will continue during the whole of the ventricular systole. The power concerned in the development of wave "*v*" being so much greater than that developed by the auricular systole, the magnitude of wave "*v*" should be as much greater than that of wave "*a*."

Wave "*c*." This wave corresponds to the pulse in the arteries at the root of the neck; it is generated by the left ventricle at the moment of the forcing of the aortic valve. The force which generates it is by far the most powerful of the forces operating at the root of the neck and appearing as pulsations. Its wave, therefore, should be the most pronounced of the three. True, it acts from without upon the vein, not from

[1] Mackenzie, op. cit., p. 149. Mackenzie attributes this wave to the gathering of blood in the auricle "during the time of the ventricular systole," i.e. the period of closure of the tricuspid valve; but this gathering takes place so quietly and evenly, under the low pressure which obtains in the great veins as these unload themselves, that it is difficult to comprehend how anything of the nature of a wave could be so produced.

within, but the great vessels, arteries and vein, are in such close contact that this should make no difference.[1] That which is difficult to be certain of is, whether the pulsation which the receiver of the polygraph transmits is a direct pulsation from the artery itself or from the vein as an intermediary, or whether it is a combined pulsation derived from both vessels. In any case it should be an energetic pulsation. Another point concerning this pulsation is its time: it is ventricular systolic, of course, but it is not *early* ventricular systolic, or, rather, not *so early* as the driving back of the *a–v* valve; for the intra-ventricular pressure has to be worked up until it exceeds the aortic pressure, before the aortic valve can be forced. The "*c*" wave must there succeed the onset of the "*v*" wave in time. No account is taken here of the slight delay of the "*c*" pulsation in reaching the neck, which would further delay its appearance.

To sum up the relationship of these three waves, "*a*," "*v*," and "*c*": judged by the energies expended in their development, the order of magnitude should be an ascending one—"*a*," the weakest wave; "*v*," decidedly stronger; "*c*," by far the strongest. It is, however, difficult to place "*v*" and "*c*" according to this criterion alone, for the wave "*v*" is likely to be reinforced more or less by actual reflux, which would accen-

[1] From the upper border of the thyroid cartilage to its termination, it (the internal jugular) "runs along the outer side of the common carotid artery, being contained in the same sheath with it and the vagus nerve, but separated from the structures by a distinct septum. The vein generally overlaps the artery in front. . . ." (Morris's *Human Anatomy*, 1907, p. 663.) See also Mackenzie, figure on p. 147.

tuate it; it will therefore be best to say simply that "*a*" should be markedly weaker than either "*v*" or "*c*."

Proceeding to the relationship of these same waves, judged now by their placing in time, the order of sequence should be: "*a*" first; "*v*" second; "*c*" third; "*v*" and "*c*" both ventricular systolic, but the onset of "*v*" preceding definitely the onset of "*c*"; the former being early ventricular systolic, the latter some appreciable time later, probably at about the middle of the ventricular systole.

Turning now to the tracings,[1] what do we note? On p. 149 the jugular tracing shows "*a*" by far in excess of either "*v*" or "*c*"; the last-named, "*c*," appearing as little more than a notch on the descending line of the "*a*" wave; the "*v*" wave, more clearly marked than the "*c*" wave, but its crest at a slightly lower level. The same thing is seen on p. 191, but there in three instances "*a*" and "*c*" are almost fused; still a slight notch on the descending line of this compound wave indicates, I suppose, the position of the carotid crest.

On p. 193, the size of the three waves is almost equal.

On p. 196, the tracing shows certain extra-systoles, but in all the systoles, normal and extra, the "*c*" wave predominates very distinctly.

In the chapter on "Some Rare Forms of Cardiac Irregularity" we find, on p. 206, that "*a*" and

[1] The references here are all to the third edition of Sir James Mackenzie's work, 1913.

"*v*" are now at times blended into one wave; here "*c*" is on the whole the maximal wave. On p. 207 of the same chapter (case of paroxysmal tachycardia), "*a*" is by far the maximal pulsation, especially during the paroxysms, and "*c*" appears now as a notch on the *up* gradient of "*a*," whilst (during the paroxysm) "*v*" disappears altogether. On p. 213 (case of auricular fibrillation) we have, in the two lower figures, the radial tracing compared with the liver tracing. Of course no carotid wave appears here, and the curve is simplicity itself, showing (before the advent of the fibrillation) waves "*a*" and "*v*" in regular sequence, and no other undulation to obscure things. After fibrillation has set in, the auricular systolic wave disappears completely and there is only the "*v*" wave, which is almost certainly a wave of reflux. I quote this case to show that the two waves "*a*" and "*v*" in the upper tracing have, practically, an identical value, i.e. that the auricular systole makes the liver pulsate as powerfully as the ventricular drive, reinforced almost certainly by a large wave of reflux from the right ventricle; this is difficult of comprehension.

In these tracings, leaving aside the question of magnitude of wave, the general order of sequence shows the wave "*c*" interposed between "*a*" and "*v*"; now this does not accord with the order of sequence of the events which produce the waves; inasmuch as the ventricular systole must produce its backward effect upon the auriculo-ventricular valve, i.e. towards the auricle, *before* it produces its forward effect upon the semilunar valves of the pulmonary artery and aorta.

Thus these tracings, as interpreted at present, do not show us conformity between the magnitude and time relations of waves "*a*," "*v*," and "*c*," and the magnitude and time relations of the forces which are credited with their production. This being so; and considering that disease differentiates between the two sides of the heart, spending the greater part of its force upon the left side, with consequent reactions (compensatory) which are not shared by the right side; and, further, that these left-sided reactions do not figure in the venous tracings of the jugular vein or, indeed, anywhere upon the surface of the body; must we not conclude that the current interpretation of the tracings in question calls for reconsideration? Clearly this will be the next step if the foregoing objections are valid; for after all they (the tracings) are facts, and as such cannot be ignored; the whole question consequently is one of their true interpretation and their availability for the purposes of practical medicine.

On this subject, generally, the interpretation of instrumental records, something remains to be said and certain caveats must be entered. In the first place it must be remembered that no mechanical recording apparatus is perfect; it cannot follow perfectly the movements it is designed to demonstrate. And why?—because it is constrained by fundamental laws which govern its own construction. Thus the law of inertia, which governs the movements of all material bodies, governs of necessity the movements of the lever which inscribes the tracing, as it does likewise the movements of the stretched

elastic membrane which receives the impulses to be recorded and passes them on to the lever-system. According to this law, the movements both of the lever-pen and of the receiving membrane will not cease with the cessation of the impulse which has started those movements, but will tend to persist until some other force comes into play to check them. The pen will thus continue in the direction of its movement until gravity steps in and controls the movement; in like manner the membrane will tend to oscillate until its elasticity extinguishes the force of the impulse. If now a fresh impulse be imparted to the membrane before the previous oscillation has had time to subside, the new impulse will compound with what remains of the foregoing and a composite undulation will result. Should a succession of impulses obtain, and the sequence be regular in force and time and the intervals sufficiently short, an average oscillatory movement of the membrane will ensue, but it will be of the nature of a composite photograph in presenting the resultant of more or fewer discrete impulses. In the case of the polygraph the skin of the neck, which is kept on the stretch over the mouth of the receiver,[1] is such a membrane, and when we consider that this membrane is under the influence of three main impulses, viz. the systoles of the right auricle and ventricle (both intrinsic), and, in addition, the systole of the left ventricle (extrinsic, via the arteries); that these impulses differ in their magnitude and that they follow in quick succession, each group

[1] We should note that this stretch cannot be kept quite uniform since it is maintained by the grasp of the hand.

of three succeeding at intervals which vary from about one second in health, to one-half or one-quarter of a second, or even less, in disease—we shall realize how intricate is the problem of the analysis of their composite oscillation into its component parts, even though we take the receiving membrane alone into consideration. But there is besides the inertia of the lever-system, which multiplies the error of the oscillating membrane, and thereby falsifies still further the record of the tracing.

That these objections are not of the nature of mere quibbling or hypercriticism will be made evident by instancing a case in point. For quite a number of years physiologists have taught that a negative pressure was generated within the ventricle, during its diastole, by the act of muscular relaxation; and this on the strength of certain curves obtained by a manometer introduced into the cavity of the ventricle. The *prima facie* likelihood of such a result, however intricate the arrangement of the cardiac muscular fibres might be, seemed too far to seek; but there were the tracings, there was Nature's own record, with a little help from science; what more was there to be said? Yet at the present moment how do we stand? Gone utterly is this teaching, for the simple reason that a newer method of recording has been devised, which has reduced to a negligible minimum the former instrumental errors due to the inertia of the parts of the apparatus: diastolic intra-ventricular negative pressure has ceased to be.[1]

Such happenings yield no occasion for despair

[1] Starling's *Physiology*, 3rd ed., 1920.

or recrimination; we have here no *opprobrium scientiæ*, but simply a record of an occurrence incidental to all instrumental investigations, a record that we advance by stumbling, and that the stumbling is, in a sense, an almost necessary stage in the advance, in that the pitfalls which beset the way of the investigator are thereby disclosed. Hippocrates has told us that Art is long and Experiment hazardous and Judgment difficult; it is so. Yet, in spite of it all, in spite also of the brevity of Life, we must make trials, and take risks, and form judgments—proving and disproving and stumbling *forwards*.

Supposing, then, it should turn out that tracings, upon which we had sought to rely, called for revision, perhaps correction or even rejection of the interpretations reached, we should still be deeply indebted to the investigator who had had the courage to seek information along a new path. For if it should become necessary to retrace one's steps, that is the way of all exploration, and the way which leads up to the final advance. It has been said "supposing," for it may be that the error is all on the side of the critic.

## CHAPTER V

### THE HEART AS AN INDEPENDENT, AUTOMATIC MECHANISM—TONE OF THE HEART

THUS far the heart has been considered in detail rather than as a whole, in dissociation rather than in association; let us now turn to this latter aspect.

The first thing which strikes the observer of the heart in action is its extraordinary self-dependence and poise, i.e. balance; together these constitute its automatism in its completeness and stability: this is most marked in the lower animals, especially the cold-blooded. Thus in the case of the frog which has been pithed, and thereby rendered insensitive, we observe the heart working vigorously and regularly, the contractions of sinus, auricles, and ventricle following each other in unbroken series for indefinite and often prolonged periods; yet here all communication with the rest of the body, via the central nervous system, cerebral and spinal, has been severed. Let the experiment proceed further by excising the heart of this insensitive and systemically inanimate animal and tying it on to the cannula of a Roy-apparatus; let it then be supplied with an artificial blood. This experiment as usually performed carries the ligature round the heart at the auriculo-ventricular sulcus, and in so doing blocks effectively all com-

munication between auricles and ventricle. Such heart is not only isolated from every other part of the body with which it was previously associated, but its very integrity has been invaded, for the heart in its totality consists of sinus, auricles and ventricle, and now the working part has been reduced to the ventricle alone. Yet what do we witness ? In general we note that there is a period of indefinite length during which the ventricle is at a standstill (" Stannius'd "). Next we observe the sudden irruption of contractions, which often arise *ab initio* in their fullness and continue thence onwards, maybe for two or three hours together, as well-executed acts, regular in force and rhythm. This spontaneous series of systoles has arisen out of the interaction of the artificial blood supply and the residual vitality of the ventricular tissues ; there are no other factors to consider.

Now, however difficult it may be in the lowest forms of life to discover two factors in the act of contraction corresponding structurally to two distinct forms of tissue, the one generating stimuli, the other responding to the excitation, there is no denying that this differentiation appears quite early in the ascending scale of life. In the frog there is a well-developed nervous system, and when we see, as just described, the sudden arising of a series of contractions in a completely isolated ventricle, we are forced to conclude that within this ventricle there must be the equivalent of a nervous structure which not only generates stimuli, but distributes them so as to bring into harmonious co-operation a complicated system of fibres.

Where is this mechanism situated ? Apparently in the proximity of the base, near the sulcus ; for if we carry the experiment a step further, by placing the ligature well below the *a–v* sulcus, and letting it bite firmly into ventricle, so as to cut off any stimuli arising at or near the base, say in the upper third of the ventricle, we then are likely to obtain a more permanent arrest of spontaneous contractions, though upon direct electrical stimulation below the ligature there is a contraction for each stimulus. But though this result is more likely to obtain, it is by no means invariable, for in relatively infrequent cases a spontaneous series of beats may arise in the part below such ligature, showing beyond all doubt the deeply fixed character of the ventricular automatism.

In the higher vertebrates and in man we get similar evidence of the stability of the automatism of the heart, though the greater complexity of structure, which a higher development produces, involves a reduction of stability. As an instance of the independence of the heart *as a whole* we may cite the effect of general anæsthesia, as by chloroform. In such case the functions of the central nervous system are abolished progressively until systemic life is reduced practically to the working of the respiratory and circulatory apparatus. True, the connexion with the centres in the fourth ventricle is maintained, and therefore to some extent control through these centres must be present; but the abolition of all the higher powers of the cerebrum, as well as of the reflexes generally, makes the presumption almost certain

that the circulatory and respiratory central control is likewise markedly impaired. In the continuance of the circulation under these circumstances of relative isolation we should have the counterpart of the circulation in the pithed frog. For evidence of the independence of the ventricle itself we must go to human pathology, instancing those cases in which the auricle is disconnected and presumably supplies no stimulus from the sino-auricular node; of course the connexions with the central nervous system are in this case intact, so far as we know, but in other respects this experiment, which disease makes, is the equivalent of the ligature placed at the auriculo-ventricular sulcus.

Experiment has shown us further that a ligature placed at the junction of the sinus and auricles of the frog-heart will bring to a standstill the parts below the ligature, auricles and ventricle, whilst the sinus continues to beat; and that a second ligature applied now in the auriculo-ventricular sulcus will by its irritation start contractions in both auricle and ventricle, but *that the rhythm of each isolated pulsating chamber is different.*[1] This experiment proves that each chamber possesses the wherewithal of an independent automatism. The frog-heart, therefore, presents a triple automatism, which in the normal circulation is so co-ordinated that the pulsations of the three chambers co-operate.

The heart in man and the higher vertebrates starts as a simple tube endowed with contractility which takes the form of a peristalsis. In the course of development the final stage shows

[1] *Practical Physiology*, 1910, ed. by M. S. Pembrey, pp. 100–102.

us a two-chambered organ, auriculo-ventricular, which a septum has converted into four compartments. Here we miss the sinus of the frog-heart, which appears to have been absorbed into the walls of the auricle. The human and higher vertebrate heart, therefore, shows us a twofold automatism only, but co-ordination of this automatism is none the less essential to co-operation.

What is this order? The cardiac cycle shows two phases recognizable by the finger upon the pulse, the one marked by an impulse, the other by a pause. The impulse corresponds to the ventricular systole, though it does not measure

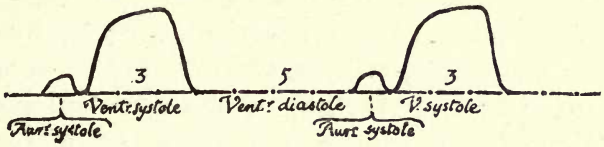


FIG. 23.—Diagram showing the time relations of auricle and ventricle in systole and diastole.

it; the pause corresponds to the ventricular diastole, but again does not measure it. To obtain measurements instrumental records are necessary. Immediately before the ventricular systole the auricle contracts; the recognition of this auricular action also requires instrumental record. Such a record would give a diagram as shown, and this is accurate in respect of the time relations, but in respect of the relative quantities of force developed by the auricle and ventricle it is quite arbitrary. However, even as it stands the diagram will serve to indicate the enormous preponderance of the ventricular systole over the auricular, though it is probable that it

greatly overestimates the auricular power-contribution. It will be noted that the auricle, after its small effort, rests for seven-tenths of a second, whereas the ventricle, after its great output, rests for five-tenths only.

In this order, which is invariable, is there any reason for the immediate juxtaposition of the auricular systole to the ventricular? Two reasons can be advanced: (1) that towards the end of the ventricular diastole the relative plus pressure in the great veins will have about exhausted itself in filling the relaxed ventricle, and that to distend the ventricle actively and complete its load the dwindling stretch of the great veins must be reinforced; (2) that the sudden stretch of the ventricular walls, by the auricular systole, will serve probably as a part-incentive to the starting of the ventricular contraction, in the same way that the increased stretch of the extensor quadriceps of the thigh excites to contraction by means of the tap on the patellar tendon. This subject has been touched upon previously.

Whence is this order, what is its structural substratum? This question has been long in the answering, but at last diligent search has discovered a peculiar form of tissue embedded in the walls of the auricle and ventricle to which must be assigned the generation of the stimuli which initiate contraction in the two chambers and determine its order. This tissue presents as its chief structural features two nodes and a bundle or trunk which breaks up into two chief ramifying branchings, distributed in either ven-

tricle, viz. the sino-auricular and auriculo-ventricular nodes and the *a–v* bundle with its branchings. This system has received great attention of late, and it is not necessary here to dilate upon it; but one may perhaps comment that, however peculiar the minute structure of the tissue in question, its function is purely nervous, i.e. it generates and distributes stimuli; and that, though it is embedded in the walls of the heart, there seems no *a priori* reason why it should not lie outside these walls like any other system composed of nerve gangla and nerve trunk; and that, though it is the means of exciting the chambers to action, it is no part of the function of those chambers any more than the nerve fibres which convey the stimulus to contraction into the fibres of the biceps muscle can be considered as belonging to the function of the biceps. The function of any organ or part belongs to the cells proper of that organ or part, and the cells proper of the auricle and ventricle are the muscular fibres which compose their walls; each chamber can contract and relax; it can do nothing else. The sino-auricular nodal system stands, therefore, apart as a separate system; it starts; it determines the order of sequence; it makes the pace of the contractile rhythm of auricle and ventricle.

The older observers had recorded that when the heart dies the last part to cease to function is to be found in the auricle, whence it was named the *ultimum moriens*. A little more patient thought, a little more perspicacity might perhaps have led us to look for the *primum mobile* within the *ultimum moriens*: it was not so, but at last the

discovery has been made that the auricular contraction which starts the cardiac cycle begins at the sino-auricular node near the point of entry of the superior cava into the right auricle. The reason for dwelling upon this matter is that, because of the priority of action of the auricle, an exaggerated importance has been attributed to this chamber; it seemed almost as though precedence signified importance. We all know that Juno would have claimed precedence on this very ground, her excelling dignity:

"Ast ego quæ Divum incedo regina . . ."

but we also know that in the procession the herald is wont to precede the potentate, and that the master of the ceremonies determines the order of procedure. Well, this same master decreed that the weak function of the auricle should come into the cardiac cycle at that point exactly at which its feebleness should be most advantageously utilized to the reinforcement of the dwindling venous pressure, and perhaps to the furnishing of a stimulus to the slack ventricle, viz. at the end of the pause; and that the instrument of the decree should be the sino-auricular nodal system.

The great act of the heart is the ventricular systole; everything leads up to and is subservient to this, and upon this act attention must be centred so as to detect at the very earliest the smallest hint of its defection. It may be that we shall get this earliest warning by watching the auricular action, and that the modern methods of investigation may prove of the greatest value in furthering this objective. These investigations

cannot then be too thorough, but as practical physicians, we must never forget that *per se* it is a small thing to us relatively whether the auricle fail or not, provided the ventricle hold out, for we know that the intact ventricle is able to carry on a working circulation, in the absence of the auricular systole. Therefore all our auricular findings must be controlled by the ventricular efficiency, and this to a great extent we can determine by direct and more simple methods, which, moreover, have regard to the state of the patient as a whole; remembering that we shall judge the efficiency of the heart by the sufficiency of the circulation.

On the other hand, the heart may be about to fail in the apparent plenitude of its vigour; such may be the aspect of things, and if at such time we could obtain, by means of a polygraphic pulse tracing or the electrocardiogram, or any other means, trustworthy evidence of the breakdown *in prospect*, the gain would be or might be enormous. I allude to cases where the heart is known to be unsound yet is showing no signals of distress, cases that we are wont to describe as compensated; for we shall rarely have the opportunity of examining people who are in seeming full health and have no reason to think that there is present any weak spot. Few will come, at the risk of their present happiness, to probe the obscurities beneath the surface; and it is best they should not.

The muscular fibres of the auricle represent the functionally active part of the walls of this chamber. These fibres cease abruptly at the

auriculo-ventricular fibrous ring. There is thus no continuity between the functional part of the auricle and that of the ventricle. No disease of the auricular fibres can cross the fibrous ring—there can be no spread from auricular fibre to ventricular fibre: the two chambers are quite distinct. Accordingly no state of flutter or of fibrillation can spread, by continuity of muscular tissue, from auricle to ventricle. On the other hand, there is a bridge connecting the two chambers composed of a peculiar tissue, viz. the auriculo-ventricular bundle; and it is this bridge which, besides conveying the stimuli which co-ordinate the individual fibres of the ventricle, links up auricle and ventricle in orderly sequence of contraction.

We know that the heart develops from a simple tube, and that the nature of the contractions of that tube at the start is peristaltic in character; also that in the sequence of things the final mode of contraction is but a modified peristalsis. We know also that in any given segment of a tube showing peristalsis the order of contraction is a fixed one; thus if we take a section of the bowel working normally, the first part to contract is the proximal part, and this contraction holds on until the next part, distally, has taken up the contraction: then the first can relax. Thus the process continues and the contents of the bowel are kept moving in one direction. In any such peristalsis not only is there a regulation of sequence, but also a regulation of pace, the rate of which must conform itself approximately to the rate of contraction of the proximal part. The proximal part not only takes precedence in time, but it

also determines the pace. Does this mode of action bestow an extra dignity or importance upon the proximal part; does it, indeed, belong at all to the muscular system of the bowel? Clearly not; it is dependent upon another system altogether, viz. the nervous supply to the bowel. The like holds for the heart, in which organ the auricle stands as the proximal part (taking into account the direction of the circulation). The function of this same auricle, i.e. contraction, is determined alike as to force, as to sequence in time, and as to pace, by a distinct system, the sino-auricular nodal system, the part played by which is essentially nervous; it comes, then, to this, that the auricle does not order, but is ordered, and that it should take rank solely according to its power-contribution in the dynamics of the circulation.

The structural substratum, then, of the heart's automatism will be twofold, a force-generating tissue—namely, the muscular fibre system—and a stimuli-generating system, which excites and regulates the force-output of the muscle fibres—namely, the sino-auricular nodal system; and when we see evidence of the spread of disorder from the auricle to the ventricle, we conclude that this spread is from within and along the *s–a* nodal system, because this is the tissue which alone makes continuity between auricle and ventricle.

This aspect of things will not conflict in any way, so far as I can see, with the very interesting and important investigations upon the intimate nature of auricular flutter and auricular fibrillation, which Dr. Thomas Lewis has communicated

to us in his recent lectures at the Royal College of Physicians,[1] for these investigations, as I understand them, consider alone the modes of response of the auricular fibres to modes of stimulation, in particular to varying rates of stimulation; they do not regard the source of the stimuli.

It is possible that the automatism of the heart, as it works within the intact body, depends, in part, upon influences which reach it via the central and sympathetic nervous systems; but that, in the main, it depends upon its own intrinsic apparatus, the *s–a* nodal system, we must conclude, because experiment shows the persistence of the automatism after all such channels of communication have been severed. This, of course, does not deny that the automatism may be influenced in every degree through the extrinsic channels, even to the violent interruption of all action; this, however, opens up another aspect, which must be postponed for the moment in order that we may consider another feature of the heart in action, which itself is very probably, in the main, an intrinsic manifestation, viz. the tone of the heart.

## TONE OF THE HEART

What do we understand by the word "tone"? It is often used rather at random without any clear consciousness of the exact meaning it bears; yet its meaning is quite real, and no less real is the part played by tone in the functioning

[1] April 1921.

of the heart and vessels. Perhaps the nearest equivalent is the word "elasticity," by which we mean that all bodies, animate and inanimate, in greater or less degree, show a property whereby they resist change of form or volume, whether by compression or extension, and tend, upon removal of the disturbing force, to return to their previous form and volume. Among inanimate objects such property is exhibited in the temper of steel or the elasticity of the billiard ball, and among things animate by the tone of the living muscle fibre.

Elasticity, tone, are not invariable quantities; on the contrary, they are constantly changing, being subject to a variety of conditions, e.g. dryness and moisture, heat and cold, and without doubt they are modified by electric states; but at any given moment they have fixed values. Tone, as we know it in the living body, is essentially related to metabolism, i.e. chemical activity; and we assume, with reason surely, that the normal tone of muscle fibre corresponds to the normal plane of metabolism of health. The same may be said of every other tissue, however low it stand in the scale of endowment, such, for instance, as fibrous tissue.

Tone varies with age. This is very striking if we contrast the firm rounded muscles of youth and the earlier years of adult life with the flabby pendulous muscles of advanced years. The lack of tone in age is exhibited in every part of the body: look at the inelastic wrinkled skin, the drooping of the lower eyelids and of the lower lip, the pendulous lower abdomen and the loose gluteal folds. And as loss of tone shows itself

externally in the character of the surface lines, so within the body it declares itself in the loss of power of the muscular fibres as seen in the relatively slow, infirm and unsteady movements of age when compared with the quick strong precision of the tense muscles of youth. To the loss of muscular tone in old age must also be attributed in large part the tendency to stasis within the hollow viscera, as seen in the constipation of the bowels and the residual urine of the atonic bladder; and, as we should expect, the senile heart gives like evidence in its tendency to dilate and its failure to empty itself in systole. Doubtless the actual weakening of the act of contraction shares in the responsibility for these manifestations, but it is certain that the loss of elasticity in the muscle fibre *whilst at rest* is also causal, and it is not improbable that it is chiefly so.

Turning from the general to the particular, let us regard for a moment the senile heart, and inquire how and where the loss of tone of the resting muscles tells specially. We know that in respect of the ventricle the cardiac cycle is occupied by systole during three-eighths of its length, and that the diastole occupies the remaining five-eighths. We know also that, though the diastolic ventricle fills under comparatively low powers—namely, the small relatively plus pressures within the great veins reinforced at the end of the diastole by the relatively weak contraction of the auricle—yet that these powers suffice. The stretch of the ventricle, as it fills, opposes these powers, and it is obvious that, as the elasticity of the fibres becomes impaired, so

the filling powers, being less opposed, must be more effective and over-distension will result. The ventricle of the senile heart, therefore, tends to become over-distended whilst at the same time its actual power to contract diminishes. This is characteristic and contributes an actual danger, which belongs to old age, viz. the danger of syncope; it also explains the increasing shortness of breath of age and the potency of small factors to embarrass the heart's action. In brief, the senile heart tends to become an overloaded organ, and one that is easily embarrassed.

Passing to the antechamber, the same holds and for the same reasons; the argument, indeed, has even more force, because the systole of the auricle occupies only one-eighth of the cardiac cycle, leaving seven-eighths for the diastole, during which it is passively yielding to the intravenous pressures, and yielding in excess because, *ex hypothesi*, the walls have lost their tone: the auricle therefore becomes over-distended. From the auricle the same tendency spreads to the veins, being most evident in the larger and medium-sized veins, for lack of tone characterizes these vessels also as age increases. We shall hence be prepared to hear that, if inflammatory movements arise in the aged, they are apt to take on a hypostatic, i.e. passive congestive, type: this is specially noticeable in the lungs.

The moral of these reflections is the danger which the period of diastole presents when the tone of the heart has been impaired. Krehl[1]

[1] Abhandl. d. K. S. Ges. d. Wiss., *Mathemat. Phys. Classe*, Bd. 17, p. 357.

draws attention to the peril of the overfilling of the ventricles, in particular the right, during diastole, and states that experiment as well as clinical observation show that the ventricle may be unprepared to deal with such overloading; I do not find, however, that he here makes any reference to loss of tone as an important factor. The records of sudden death in aortic regurgitant disease furnish striking illustration of this subject; in this case the surcharging is of the left ventricle, and it is the more perilous because the relaxed chamber fills by reflux under great pressure, the greater by reason of the marked hypertrophy of the left ventricle, and its powerful systole. In this case we may have no need to invoke loss of tone to explain things, though it is evident that its presence would accentuate the danger.

We have seen that the automatism of the heart presents us with two structural factors, the one force-containing—this is the muscular fibre system; the other endowed with the property of generating stimuli, which are capable of converting the potential of the fibre into dynamic energy, and of so distributing the stimuli as to bring into active co-operation the host of fibres which constitute the walls of the heart—this is the sino-auricular nodal system. These two factors suffice to account for the sequence of systoles which the living, working heart exhibits. Of what nature, then, is tone? Does it belong to the one or to the other, or to both? Without doubt it also is a product of the interaction of these factors, and that

just as in the case of the skeletal muscle the tone of the fibres depends upon the integrity of that part of the spinal cord which furnishes its nerve supply,[1] this implying an interaction between nerve cell and muscle fibre, so in the case of the heart the tone of the muscular fibres must depend upon a like interaction between the fibre and the sino-auricular nodal system. This interaction must be of the nature of a stimulation, which is more or less continuous, i.e. sustained, as against the interjected stimuli which yield the systolic sequence. The systoles of the heart would seem to mask, in a sense, the tone of the heart, which in the form of a sustained elastic force underlies them; the diastoles reveal the substratum upon which the ventricular strokes are superimposed. It is essential that we should discriminate between these two forms of manifestation of energy, for when we bring our medicaments to the aid of a failing or disordered Nature we must recognize a twofold objective, viz. on the one hand the need to modify the force of each systole, reinforcing or reducing it; to regulate the sequence of the beats, equalizing their force and intervals; to accelerate or diminish the frequency of the rhythm—on the other hand the call for a raising or a lowering of the tone of the muscle fibres, as the case may be. The one objective might be said to regard the systolic part of the cardiac cycle, the other to restrict itself to the diastolic element, but this would be likely to lead astray, for though the systole may mask tone, it does not

[1] Cf. Gowers, *Diseases of Nervous System*, 1886, vol. i. "Discussion on the nature of the tendon reflexes"; see in particular p. 17.

abolish it, and tone must apply to the whole cardiac cycle. It is probable that tone and systolic force move more or less together.

Tone is present in the heart, but not prominently to the fore; whereas in the vessels, especially the arteries, tone is in striking evidence and a force to be reckoned with by the heart. Tone, as it is manifested in the arterioles and smaller arteries, presents the mechanism whereby a resistance is thrown into the vascular circuit—here, there, or generally. If restricted to a small vascular district, its concern is strictly local, in its regulation of the flow through that district; but if it apply to a great vascular area, *a fortiori* if it become general, the welfare of the whole system is at stake, and it is then that the heart has to measure itself against its great antagonist, it is then that the distinction between tone and the systolic powers of the heart becomes unmistakable. Thus if the arterioles generally contract, the systoles of the ventricles must be reinforced in proportion, and the conflict of the two will beat up a blood pressure which is adequate to force the arterial narrows and maintain an effective circulation: the heart is then triumphant. Should, however, the ventricles not prove equal to the occasion, the reverse obtains. In contrast with this active opposition of tone to systole, we have, in its extreme form of withdrawal, the toneless vessels of the state of collapse, in which condition the vascular resistance of the circuit is so reduced that the unopposed systoles are powerless to beat up a blood pressure of any value. We glimpse in these examples of the part played by tone what an all-powerful factor it may prove

itself, whether by excess or default; and that in its midway expression it furnishes that degree of antagonism to the contractile forces of the heart which is necessary to the circulation of health. Without this action and reaction life could not be; it is the fundamental basis of all cosmic physical activities.

To revert to the heart, it presents to the incidence of the drug two elements, the muscular fibre and the *s–a* nodal system, and the drug is capable of effecting an improvement of the tone of the heart as also an improvement of the beat. Typical of the former class of remedy are such drugs as pituitrin and physostigmine; typical of the latter class, such drugs as the diffusible stimulants, e.g. ether and ammonia, and we may add camphor, on account of its volatility: among other remedies, whose influence seems to fall principally upon the mechanism of the beat, are the caffeine preparations, especially the sodio-salicylate and benzoate, and strychnine. Hypodermic injection of all these remedies gives the most speedy and powerful effects.

In respect of drugs of the pituitrin class, the incidence is not confined to the heart; the circular fibres of the arterioles feel the effects as well, and it will be difficult, perhaps impossible, to apportion these effects; it must suffice that an improved cardiac and vascular tone is recorded. But we must once again lay stress upon the nature of the cardiac effect, which tells principally upon the heart during diastole and, in virtue of the increased tone effected, obviates the danger of overloading.

Upon the list of cardio-vascular remedies, where do digitalis and its congeners come? Do they act mainly by improving the tone of the heart, or by reinforcing and regulating the beat? Without doubt they secure both these objectives. Their action upon cardio-vascular tone can scarcely be denied, and prominent in this action is the direct effect upon the vessels. This was shown very strikingly in experiments performed by Dr. Ringer and the writer upon the peripheral circulation of the tortoise (after pithing); and Schmiedeberg was wont to attribute the chief cardiac effects of digitalis to the rise in blood pressure caused by the contraction of the arterioles. This is a complicated question and can hardly be raised here; we must be content to accept increased cardio-vascular tone as the result of digitalis action. There is, however, one digitalis effect which seems purely cardiac, viz. its power to regulate disorderly action; in certain cases of this type its action seems almost specific. This co-ordinating effect is surely nervous in its nature, and though it may be brought about in part through extrinsic influences conveyed to the heart, as by the vagi nerves, is it not more likely that in the main it results from influence upon and through the intrinsic co-ordinating apparatus of the *s–a* nodal system? And with reference to this it may be pointed out that this apparatus lies very close to the inner surface of the heart, in fact just below the endocardium, and that so placed it would be accessible to direct stimulation from within the chambers of the heart as the blood circulated through the organ. This mode of

action has been discussed in a previous chapter, when treating of the utilization of the blood stream as a carrier of remedies, and it was pointed out that such direct action was likely, especially with the strong diffusible stimulants, such as ether, ammonia, camphor; in the case of digitalis it is clear that direct action across the endocardium is much less probable owing to the rather sluggish physico-chemical behaviour of the glucosides, to which class its active principles belong. In confirmation of this we shall remember that digitalis tends to develop its action slowly.

To conclude this therapeutic digression, it should be advisable in all forms of heart failure to combine in our treatment remedies calculated to reinforce and, if need be, regularize the beat, along with remedies calculated to raise the tone of heart and vessels. This, not necessarily by combination in the same dose, but rather by separate administration at intervals in the course of the day. Here we shall note that the effect upon tone, especially vascular tone, is more persistent than the effect upon the beat of the heart, and accordingly that the need for repetition of the stimulation is greater in the case of the drug which reinforces the beat than in that of the tone-raising agent; thus in combining the use of pituitrin or physostigmine with, say, strychnine or caffeine or camphor, the latter will require more frequent administration.

## CHAPTER VI

### THE HEART IN RELATION TO THE BODY AS A WHOLE

*Ut pulsor, sic pulso*

THE last aspect of the heart takes cognizance of it as the central feature of the circulatory system, and shows how, by virtue of its innumerable vascular dependencies, the capillary areas, it enters into intimate relation with the life of every single part of the body. The heart and its vessels are now to be regarded as one whole, in which these two stand as part and counterpart, the heart acting, the vessels reacting, and *vice versa*. Between this action and reaction the vital fluid, the blood, flowing in an even stream, bathes every tissue cell, supplying its necessities nutritional and purificatory.

The vascular system, heart and vessels, is covered by an interlacement of branching nerve fibrils, with interspersed ganglion cells here and there; the fibrils are the ultimate ramifications of the cerebrospinal and sympathetic systems, and by their means every individual part is brought into touch, and the entire framework of the body knit into one sentient whole. Beneath the covering interlacement of nerve fibres lie the muscular fibres of the heart and the vessels, in readiness to respond, by contraction or relaxation,

to the impulses which reach them along the lines of communication. Certain of the fibrils report upon the state of the tissue cells of the part, certain others convey the reflected response, and according to the nature of this response (we refer now to the vessels) a resistance is thrown into the circuit or is withdrawn, as the arteriole fibres contract or relax. Here is a large field of operation for Newton's law of action and reaction ; and how minutely the sentient field is mapped out is borne in upon us as we perceive the effects of a bead of matter pent up under the periosteum of the fang of a tooth or beneath the finger-nail—the resultant acute pain, accentuated by each pulse-beat, attesting the urgency of the signals of distress sent out, whilst at the same time the vascular response, by its sharp delimitation of the local afflux of blood and by the intensity of the redness, marks the precision with which the focal disturbance is located and quantitatively dealt with.

It is not, however, the intention to attempt a detailed description of the inter-relationship of the vascular and nervous systems; the utmost endeavour will be to refer to certain main lines of the plan.

We have seen the isolated heart beating automatically according to a well-established order; we now see it in relation to innumerable parts, each with a more or less individual life, and each probably or possibly in a different phase of activity, now active, now passive, in varying degree. This congeries of parts, exposed to the accidents and incidents of external forces, which are bound to impinge differently, seems to

present an insuperable obstacle to uniform action on the part of the central organ of the circulation; yet of such hindrances, in general, we see no signs, the admirable, even sequence of beats persisting. How comes this?—for though it is unthinkable that the heart should be able to alter its beat to the multitudinous calls, which must come from the periphery, and project its differences in as many directions, yet it is equally certain that the calls are there. How, then, are they met? By a most simple provision—namely, the segmental representation of the entire body in the central nervous system, according to which each separate part has a portion of the grey matter of the spinal axis allotted to it, to which is assigned the government of the vascular supply of the part. The afferent nerve conveys the call of the tissues to the local centre; here it unlocks the precise amount of energy needed, and this energy is then projected centrifugally to the focus whence the call issued. In this way the heart is relieved of all concern for the circulation of the individual vascular area.

True, but does this suffice to explain matters? for the problem as far as the heart is concerned is a mechanical one, purely and simply—it has to negotiate the sum of the resistances which the whole circuit presents, no more, no less. If, now, you alter the resistance here or there, how can you prevent the heart feeling this alteration, for it must have added to or taken from the sum total of resistances? This difficulty has been dealt with in a previous chapter, where we have explained how the anastomotic system of vessels comes to the aid of the heart by supplying relief

channels which, by dilatation or constriction, obviate the effects of the local increase or decrease of resistance in a given vascular area. In this way the *average resistance* of the whole circuit is maintained at practically the same level, and an equable delivery of blood in the unit of time ensured; it is not necessary to reopen this question more at length.[1]

These general statements hold so long as the local vascular disturbance is confined to the individual area or to scattered areas here and there, whose sum is negligible, as compared with the total vascularity of the tissues. When, however, the area involved exceeds a certain proportion of the sum total, the compensatory powers of anastomosis are unable to meet the demands upon it, and the heart, faced by a widespread obstruction, e.g. of the splanchnic area, or by an equally widespread area of relaxation, must feel the effects and must respond, if it is not to succumb to the obstruction or to the atony. In the first instance, that of the widespread obstruction, the arterioles contracting in mass upon their contents must immediately raise the blood pressure slightly, but the more appreciable rise will have to wait the more vigorous contractile response of the heart. The blood pressure will continue to rise until a sufficient supply of blood for the capillary needs is forced through the narrowed arterioles, and there it will have to remain. In the second instance, that of the widespread relaxation, the arterioles, letting go, so to speak, their grip on their contained blood, the blood pressure falls precipitately; the fall

[1] See pp. 97, 98.

is accentuated in proportion as the loss of tone of the capillaries and venules adds itself to that of the arterioles. Such is the condition which obtains in collapse, and the cardiac response demanded is a reinforced beat, accelerated or not. The dilemma which presents itself here arises from the fact that in states of collapse the delivery of blood into the heart is actually diminished, and in consequence its chambers less well filled; the urgent cry, therefore, for a raised blood pressure becomes impossible to meet, since it is impossible to distend the toneless arterial system with the actual quantity of blood at the disposal of the heart. The therapeutic needs of this case are twofold, viz. (1) the administration of a remedy of the pituitrin class to restore arterial tone and so hold up the cardiac efflux; (2) the administration of a cardiac stimulant such as caffeine, strychnine, camphor, ether, etc., to beat up the arterial blood pressure. In this order, or simultaneously, according to the urgency of the case, these remedies should be given.

Let us now pass to the consideration of the mechanism by which, in conditions such as the above, the cardiac response is brought about; this opens up the whole question of the cardiac reflexes.

The first among the reflex effects which calls for note is that which follows upon a rise of blood pressure, occasioned, say, by obstruction of the aorta or stimulation of the splanchnics; this effect is retardation of the pulse. It is not invariable, but is so frequently observed that it

has been set down as a law, the law of Marey—viz. that as the blood pressure rises, so the frequency of the heart's pulsation falls, or, otherwise stated, the pulse rate is inversely as the blood pressure. It is supposed that the path of this reflex is from the inner surface of the left ventricle and root of the aorta, whence along certain afferent lines a stimulus is carried to the heart centre in the medulla oblongata, and thence reflected along fibres in the vagus back to the heart.[1] The stimulus is rise of blood pressure, to which the chambers of the heart (like other hollow muscular viscera) are very sensitive, and in its essence it will mean muscle fibre stretch (tension). This reflex is cardio-inhibitory; and looking at it, we cannot help noting that the slower rhythm is caused in the main by a lengthening of the diastolic period of the cardiac cycle, and that so the ventricle is afforded a longer time to recover from its increased systolic efforts, necessitated by the increased resistance opposed to it; nor this alone, for similarly the physical stretch of the arteries is also given more time to relieve itself across the capillaries. The slower rhythm is therefore conservative both of muscular fibre and elastic tissue.

Another reflex is said to proceed from the venous end of the heart, viz. the right auricle; it arises in states of distension, that is, of increased intracardiac pressure. This reflex is stated to bring about an increased frequency; whether by diminishing vagus tone or by stimulating the accelerator nerve fibres is not certain.[2] These

[1] Starling, p. 1024. [2] Idem, pp. 1023, 1024.

points are of great difficulty of determination, for, besides the choice between two alternatives thus presented, we may note that the conditions of distension of the right auricle, mentioned above, involve a general overloading of the venous system, and that this means generally, if not always, an ill-filled arterial system, i.e. a lowered arterial blood pressure; this being so, and recognizing that a rise of arterial blood pressure occasions a reduction in the pulse rate, it should not be surprising if a fall in arterial blood pressure occasioned a rise in pulse rate and through the same reflex paths.

Accepting the teaching of these two reflex paths, from the arterial and venous ends of the heart respectively, we see in them examples of an extrinsic governance of the heart by a centre in the medulla oblongata through the operation of a stimulus, viz. blood pressure, working from within the heart cavities.

Leaving now the heart area, we find that there are other reflexes, which, originating as stimuli at nearly every part of the periphery, find their way by devious routes to the same heart centre in the medulla, whence they are projected upon the heart, with the result that its action is retarded (even to complete temporary arrest) or accelerated; reinforced or diminished. Thus a smart tap upon the exposed intestines of the frog may, for a while, arrest the heart altogether; but the same result may follow upon stimulation of any other part of the body provided it be sufficiently intense. Here it is worth while noting "that in general the alimentary tract appears to be in closer connexion with the cardio-

inhibitory centre than other parts of the body," and that irritation of the inflamed peritoneum is apt to " produce a marked inhibition," [1] because these observations fit in with clinical experience and find their therapeutic application.[2]

These instances of extrinsic heart governance do not exhaust the list; thus we may bring action to bear upon the heart by a direct influence of the blood, as it circulates through the medullary heart centre, whether physically, by means of an altered blood pressure in the vessels, or, chemically, by an alteration in the constituents of the blood, or, of course, through a combination of these two factors. Again, it may result from an influence exerted through the higher cerebral centres upon the heart centre. It is here in particular that the emotions figure, under the sway of which the cardiac response is so ready and so pronounced, that we cannot wonder at the older biologists, when busy domiciling the various manifestations of the ψυχή in the organs of the body, selecting the heart as the seat of the emotions.

Setting aside this allocation, as we must, it is no less essential that we recognize the readiness of the cardiac response to the psychic disturbance, and without doubt we have yet much to learn in this respect. In saying this the writer has in

[1] Foster, *Textbook of Physiology*, 5th edition, 1888, pt. i, p. 295.

[2] Disordered states of the gastro-intestinal tract are a very common source of deranged and impaired heart action, and in our treatment of this organ we must always look to the state of the alimentary system, and, as a primary duty, correct any deviation from the normal; on the other hand, we must recognize that, therapeutically, we can utilize the stomach and intestines to stimulate the heart, witness the action of the " cordial."

mind a recent case of an enlarged and much hypertrophied heart, showing pronounced signs of distress including marked arrhythmia and breathlessness, which symptoms were greatly relieved on the lifting of a severe mental depression which happened to be present and to which the patient was subject. The latter, a doctor and a close observer, had himself noted the sequence on more than one occasion. Another case, somewhat allied and also recent, was marked by a sudden acute attack of heart failure, followed by the signs of irritable heart, and attended by neurasthenic symptoms, which latter in the course of things appeared to be the dominant element in the case, and suggested that the neurasthenia was the more basal condition and the heart attack rather of the nature of an epiphenomenon, incidental to the nervous state.

Be this as it may, it should not be surprising that a general nervous depression would include the heart in its sphere of action, so lowering its tone as to accentuate an existing heart failure or predispose to its onset.

Regarded then from either point of view, spiritually or physically, the heart is open to influence and will respond, in this sense living up to the motto chosen for it on the title-page :

*Ut pulsor, sic pulso*
(As I am moved, so I beat).

Thus in the relational life of the body, the psyche or animula takes its place.

To conclude this brief outline of the extrinsic governance of the heart, it may be stated, on

experimental findings, that if the nerve tracts which establish the connexion of the heart with the rest of the body be destroyed, relational life ceases; for though systemic existence may be prolonged, even for months, it is bare existence and not life, since there can be no adaptation of the circulation to the exigencies of living,[1] in its manifold relation to outward circumstance.

[1] Starling, op. cit, p. 1012, "The Nervous Regulation of the Heart."

## CHAPTER VII

### HEART DISEASE

THE presence of heart disease does not necessarily announce itself; it may have been in possession for long periods of time without ever having given a note of warning; and so it comes about that, now and again, quite unexpectedly, in the course perhaps of a routine examination, the physician finds himself up against the unsuspected but unmistakable signs of established disease. Is it disease? There has been no dis-ease, and by no signals of any kind has the wearer of the heart been made conscious that the central organ of his circulation is out of gear. True, it has been quite latent; but it speaks plainly enough now through the stethoscope, and declares a sound which should not accompany the normal working of the heart: here is an abnormality. Does that constitute disease? Not necessarily, if by disease we are to understand a morbid process in active progressive movement; for it may be a malformation and as such may show no progressive character. But even so, though the local morbid process be arrested, we must still regard the heart as diseased if the arrested local trouble be capable of reacting disadvantageously upon the rest of the body, as, for instance, a fused valve which by its obstruction puts a strain upon the heart, though the fusion have been long stationary. Failing the evidence

of such a morbid, progressive *reaction*, we may if we like draw a distinction between the malformation and disease, though we could hardly then escape the conclusion that the malformation, being the result of a deviation from the normal at some point of time, did indicate active disease in the past. The distinction, however, between a malformation which has ceased to react adversely and true heart disease may be defended, and has its practical value. We shall thus have to distinguish in practice between the mere abnormality, which has no morbid potential, and true heart disease, as above defined. The patent foramen ovale would be an instance, in a majority of cases, of the malformation which was a mere abnormality.

Let us take another instance of the abnormal, and endeavour to gauge its true significance. We find, say, an irregular rhythm of the heart—what may it mean ? Is it the sign and the measure of active disease, or is it due to an abnormal laying down of nerve cells and nerve tracts, within the organ itself or outside it, during the developmental stages, as a result of which there is unequal generation of stimuli within the cells or faulty conduction of normally developed stimuli by certain of the nerve fibres ? Either of these defects might account for the anomalous rhythm: a third possibility would be the presence of groups of muscular fibres of unequal sensitiveness. The question must be investigated on similar lines, the points for determination being: (*a*) the stationary or progressive character of the symptom ; (*b*) the ability to trace it far back in the history of the case without finding any

evidence of a definite arising or of the genesis of a likely cause, or on the contrary the presence of positive findings in both of these respects; (*c*) the absence of any indication that the heart is under stress or the circulation impaired, as against the presence of heart distress and circulatory failure. In this way we may be able to diagnose between an innocent cardiac eccentricity and true cardiac disease; and it is important to remember that in the characters of the anomalous rhythm the former may closely simulate the latter.

The term "functional" is often applied to abnormal workings of all kinds and with special frequency to cardiac manifestations. It is customary to contrast it with the term "organic." The terminology is not very happily chosen, but it has become so established that we had better make the best of it and endeavour to make clear how it has been applied. The term "functional" has been used to describe those deviations from the normal which have, so to speak, no morbid momentum of their own, and which do not present easily demonstrable structural changes; they are in many cases secondary to other deviations, in themselves of lesser import, these latter being in general more or less amenable to treatment, and on the success of such treatment the functional derangement tends to disappear. The characteristics, then, which mark the functional perturbation are, its essential dependence upon departures from the normal, which themselves for the most part lack stability, and are therefore transitory; and the absence of structural changes which show. In contradistinction, the organic de-

viation from the normal is marked by a well-defined morbid momentum and by the presence of tissue changes, gross or minute, which can be demonstrated. The functional disturbance oscillates with its unstable cause and disappears upon its subsidence. The organic trouble, having more momentum, is more rooted and fixed and more resistant to curative forces. When all is said, however, we have no scientific finality in the distinctions drawn, for we can conceive of no functional change without a corresponding and equivalent structural modification and *vice versa.* As examples of the functional disorder we may cite the hæmic bruits so commonly present in anæmic states, which tend to disappear as the anæmia disappears and an improved general tone of body is established.

Another form of heart manifestation which we meet with is difficult to place; it cannot be described as diseased, for no precise morbid focus is discoverable; neither can it be said to be healthy, for changes of a degenerative type, affecting the cardiac tissues generally, may be shown. It cannot be said to be abnormal, for it belongs to the normal course of life, and is in a sense preparatory to the normal ending of life in its antithesis—death. This is the senile heart; the worn-out heart; the katabolic heart. Katabolism, anabolism are present from the first moment we draw our breath unto the last moment; but whereas in the earlier years and the mid-years anabolism preponderates, in those which follow katabolism is in the ascendant. The senile heart, however, if it has worn well, will

have kept its downward path at the same pace as the rest of the body, and harmony will continue, for the forces of the heart will be adequate to meet the downward grade of functional demand made upon it. Disease means discord—here is none. The heart change is part and parcel of the general change.

Unfortunately for the heart, we do not wear out equally—we are apt to be too young here, too old there. The legs and arms may have acquired a habit of activity, which their presiding nerve centres may maintain, but to which the heart and its presiding centres have not lived up. The legs may then outwalk the heart, and the possessor of the twain is brought up puzzled and discomfited by a pain, a palpitation, or a distressed breathing. There is now no longer a proper corporeal balance, and how shall we label this if not as disease? We are upon the horns of a dilemma which we can only meet by admitting that the senile heart may be both disease and not disease. It is undoubtedly disease if decay occur prematurely and preponderatingly in the heart, but if it proceed so that it keeps pace with the decay in the rest of the body it is not disease. In any case the senile heart deserves a place by itself.

What are the features which characterize this heart? It will be featureless provided that the demands made upon the heart are kept proportional to its strength; provided, in particular, that the pace be reduced to *petite vitesse*, or at any rate below the *grande vitesse* of youth. Given this reduction of rate of energy-output, the total output in the day's doings may still be quite

considerable. It is probable, as already stated, that the chief physical change presented by the senile heart is a reduced tone (elasticity) rather than a diminished contractile power (though this also must be reduced). Such a heart will tend to become overloaded with blood during the diastole because of the loss of tone, and this will put the systole at a disadvantage.

The senile heart will thus be a heart of lowered potential, restricted particularly in respect of its rate of output; it will also be easily embarrassed by external conditions which the firm elastic heart of youth might not regard. Thus alimentary tract conditions, especially flatulent distension with its upward pressure on the diaphragm, may gravely impair the cardiac activities of the aged. So much is this in evidence that the senile heart may be described as the easily embarrassed heart, and defined as such rather than as diseased, since there is no definite morbid departure, indeed no change but such as comes of usage and of time, to which all things terrestrial are subject.

No mention has been made thus far of heart states which may accompany systemic disease, and even constitute a dangerous accompaniment, yet which are never labelled heart disease. They do not even come under the heading of functional cardiac derangements, though, of course, there is functional impairment and they are in a certain sense secondary; the reason for this is that in general they may be shown, macroscopically or microscopically, to depend upon (or to be attended by) definite organic changes. Such heart states are met with in fevers and, in proportion to the degree of the pyrexia and its duration,

are wont to be accentuated. In the prolonged pyrexias the state of the heart, as it bears well or ill the raised temperature, forms one of the most important elements in the prognosis. The toxins circulating in the blood, in consequence of the infection present, together with the abnormal heat, are without doubt responsible for the changes in the walls of the heart, whether muscular fibre or nerve element be preponderatingly selected or both suffer appreciably. As examples we may instance the heart of typhoid fever and of diphtheria; the former exemplifying, probably, the effect of prolonged pyrexia, *qua* temperature, the latter the effect of the chemical poison rather, inasmuch as diphtheria does not as a rule show much pyrexia. In the same category would come the cardiac state in ulcerative endocarditis, if one could separate the condition of the heart due to the septicæmia from that due to the local cardiac lesions; and here also would be found the heart of pernicious anæmia, a pyrexial cachexia. The cardiac asthenia in Addison's disease and in phosphorus poisoning is part of a profound general depression of vitality, accompanied by marked degenerative changes, toxic in nature. For want of a better name these cardiac states might be classified under the heading—the poisoned heart. This heart is not secondary to the general state; on the contrary, it is an integral part thereof.

In the course of practice each and all of these hearts will come before us for advice and treatment: they may, perhaps, be conveniently tabulated as follows:

**Cardiac states**

- **Heart disease proper**
  - **Organic**
    - **Progressive** — Progressive both *locally* at site of lesion and *systemically* by reaction upon the whole body, and, the local disturbance being in movement, the general reaction must be so likewise.
    - **Non-progressive** — Non-progressive *locally*, but *systemically* progressive. Here will come certain forms of *malformation* arrested locally, but of sufficient magnitude to put a strain upon the heart and circulation, which strain will be an increasing one.
  - **Functional**
    - **Progressive** — The innocent stationary *malformation*.
    - **Non-progressive** — As a rule the functional disorder is a mild affection and, being secondary to trouble elsewhere, is devoid of any momentum of its own; still, the while it lasts it forms part of an existing vicious circle and so reacts harmfully. If it exist in higher degree and be maintained long it may result in definite organic changes, and will then leave its purely functional category. The heart disturbances of alimentary tract disorder may illustrate this type; also those dependent upon an impoverished blood.
- **Heart disease without separate individuality, as against heart disease proper** — The anomalous behaviour of the heart in this type of functional disorder, showing no harmful reaction upon the system, does not call for treatment. Certain forms of abnormal rhythm belong here. This heart may be named the *eccentric heart*.
- **The senile heart** — This form of heart disease is integral with disease of the whole system; it is not dependent upon this latter in the same way that the functional progressive disorder is, but is part and parcel of the whole morbid movement. It is exemplified by the heart of the febrile state and, in general, of toxæmias as a class; it may be styled the *poisoned* heart. It may call for very vigorous direct heart treatment. The heart of Graves' disease should probably be placed here.

This heart is the result of degenerative changes, yet is not necessarily an instance of disease, provided that it keep in line with the degenerative changes proceeding elsewhere; it becomes disease when it steps out of that line. It calls for guidance and maybe for definite cardiac treatment, especially if unrealized by the patient, or if, though realized, the limitations which it imposes are not accepted.

It must be repeated that disease may not announce itself, but may remain silent, both symptomatically and in respect of physical signs, even to the point of impending disaster. Atheroma of the coronary arteries and their branchings, leading up to a fatal anginal attack, will be an example of such latency. The case of Dr. Thomas Arnold, of Rugby, so graphically described in Dr. Latham's lectures on diseases of the heart, under the initials "T. A.," is an instance in point[1]; though in this particular instance, if memory serves me, coronary atheroma was not shown.[2]

What is the meaning of this latency, especially in the case of true organic disease? It signifies either that disease during a large part of its course may not interfere appreciably with the proper functions of the organ, or that, if it tend to interfere, the tendency is overcome by certain protective mechanisms which automatically come into play, e.g. the mechanism of compensatory hypertrophy. The sudden development of the signs of active disease (long latent) may be explained by the more or less sudden exhaustion of such protective mechanism.

[1] 2nd ed., vol. ii, pp. 373, 374 et seq.

[2] Having consulted the above reference again, I find that the absence of coronary atheroma is strictly true, "with the exception of a slight atheromatous deposit situate about an inch from the orifice of the artery. This, however, did not appear to diminish its cavity." But the interest of the case lies in this, that "there was but one coronary artery, and considering the size of the heart, it appeared to be of small dimensions. It with some difficulty admitted a small director." This defect may have been congenital. "T. A. was within a day of completing his forty-seventh year," and "up to a very few hours before his death, both body and mind seemed equally to give proof and promise of health."

In the foregoing table, the separation of functional from organic disease is necessary from a practical point of view, but scientifically it cannot be defended; for it is not possible to imagine any functional change without a corresponding structural modification, however difficult it be to demonstrate the change. Force and Matter constitute one inseparable unity, and just as force moulds matter, so matter exhibits force. Raise the temperature of a bar of iron, and under the inflow of the heat energy the bar expands, the distance between the individual particles of iron having increased; but, it may be argued, this is not necessarily change of structure, for the iron may be equally homogeneous. Yes, it is change of structure, for density is one feature of structure and this has changed. In a case such as this the demonstration of change of structure is simple enough, but let us take another instance, that of the soft iron core of the electromagnet, and let us subject it to the influence of an electric current circulating through the enveloping coil of wire. What do we observe? We note the sudden acquisition of magnetic properties by the iron core, which properties disappear as suddenly on shutting off the electric current. In this case the iron exhibits a new function and then loses it. Can we imagine that the molecules of the iron have remained passive under this influx and efflux of energy? It is unthinkable, and even if we could exclude material changes in density we should be forced to assume vibrational changes in the constituent particles, i.e. changes in the molecular state, changes in polarity, or however you name them;

but such changes would be in fact structural. Whether physics can demonstrate these changes of form in the iron core I do not know, but they must be there.

If this argument hold true of the phenomena which physics deals with, it will hold true of those manifestations which belong to physiology, because the physical properties of substances are maintained after their incorporation, and there will therefore be no functional change which is unaccompanied by structural change. For all this we cannot wisely dispense with the term "functional disease" as contrasted with "organic disease": it has indeed a real meaning which may perhaps be best illustrated by reverting to the analogy of the electro-magnet set for Faradic use. In that case the magnetic polarity lasts only so long as the polarizing current lasts; upon each interruption of the current the molecules of the iron core swing back to their unpolarized state, the working of the induction apparatus depending upon this very quality of reversion. Without straining analogy, fully aware of all its dangers, may we not regard the power of reverting to a previous state (of health) as defining what we have in mind when we speak of functional disease, namely, that state of the tissues in which, however marked the functional disturbance and presumably, also, the molecular changes, these latter have not become so firmly established as to be incapable of swinging back to their normal state upon the withdrawal of the disturbing factors. If this be accepted, it will follow almost necessarily that organic disease, i.e. more permanent tissue changes, may grow

out of a functional disorder, and that the likelihood of this will be proportionate to the persistence of the disturbing factors. Does not this view fit the facts of the case clinically? For do we not meet with instances of disease presenting both the physical signs and the behaviour of indubitable organic mischief, which have begun as disorders regarded as functional? In illustration we may cite certain cases of organic heart disease, as a terminal event in severe and protracted Graves' disease. And, again, do we not often find it impossible to say with any confidence whether we are dealing with a functional or an organic derangement? Take the question of the beginnings of pernicious anæmia and the doubts which beset and pursue us.

Conversely, can we ever assert dogmatically in a given case of organic mischief, even pronounced, that the power of the part to revert to a state of health, or at any rate to a state in which the morbid movement has been suppressed, more or less—can we ever state with certainty that this recuperative power has wholly disappeared? There is a loophole here, given favourable conditions, through which hope may enter though the outlook seem unpropitious, not to say ominous.

These considerations raise another aspect of biology, both normal and morbid, which applies here. In the functional disorders, so called, emotional factors or emotional manifestations are apt to be prominent, to such an extent, indeed, that the terms "emotional" and "functional" are used at times almost synonymously. What is the meaning of this? Is it that in the functional

disorder the emotional factors come then only into active operation, whilst in health they are in abeyance ? Clearly not, for the emotions constitute an integral part of human life, and they are in constant operation throughout its continuance, though in varying degrees of intensity. They are factors of great, almost incalculable, potency, so long as they are controlled and directed, and it is just here that we discover the explanation of their prominence in the functional disorder ; for, however dark the *modus oriundi*, it is in a loss of control and direction of the emotive powers that we must seek and find one of the distinguishing features of functional disease.

In normal life, then, the emotions are continuously at work, and the fullness and beneficence of life is in proportion to their intensity and their direction to worthy ends. We are confronted here with one of the most mysterious facts of life, namely, the degree in which the operations of the body, its material manifestations, are swayed by the emotions. Let a great desire, be it love or ambition, in its most personal or its most altruistic form, take possession of the ego, and what will it not accomplish ? Just as the circulation of the electric current around the iron core confers upon the iron new manifestations of power, so the emotion at work upon the vital tissues confers upon them, if not new powers, then new potentials ; bodily and mental efforts are put forth and endured incomprehensibly ; the chemistry of the metabolism is quickened, so that calories cease to have any value, for from a diet the most exiguous, life is maintained at a

maximum of output. There is no romancing in all this; it is a plain matter of recorded fact.

But if this be so, how much it behoves us as practical physicians to study this aspect of humanity—the man with an ideal, some desirable objective, in view or to be implanted; for though this does not concern us medically in the ordinary way of life, but belongs rather to the parent, the instructor, the director religious or ethical, it becomes very much our concern in sickness, since the same potentials, which the emotions confer so fruitfully upon the healthy body, may be enlisted to combat the forces of disease. Here is another reason for caution in forecasting the course of disease and delivering ultimatums, namely, that we cannot tell to what extent the life in question is capable of being charged emotionally with the desire to live, with self-confidence and the will to endeavour—the problem, as a first step, is to find the incentive. We may recall the anecdote told by Sir William Jenner, quoted elsewhere, of the iron master, approached by his doctors in consultation, and informed that his days were numbered: "What! die when pig-iron is so much a ton? Never!" —and he went on living.

On the other hand, whilst recognizing to the full the potentiality of the emotions for good, it must follow of necessity that the withdrawal of these vitalizing powers should be succeeded by a corresponding devitalization of the system; and this is exactly what we do observe. The devitalization will be in precise proportion to the intensity of action which the withdrawn incentive had been previously exerting, and it may proceed

to such lengths that unless we are able to break in upon the indifference to life which has arisen, or even the actual desire that death should make an end to a vacuous existence, an end there will be. The term " broken heart " is often applied to such modes of dying; it derived from the days when the heart was regarded as the seat of the emotions. Now we know that, wherever else they may reside, it is not within the chambers of the heart, responsive as this organ is to their play ; but in spite of this knowledge the metaphor must be allowed, for the heart as the symbol of the emotions is too firmly incorporated in our language to be dispossessed. We shall, however, know how to interpret.

The applicability of these considerations is to the whole body of morbid manifestations, but, inasmuch as diagnosis must ever precede treatment, it remains that a warning be entered here in respect of the investigation of disease. In diagnosis we are dependent upon two sources of information; the one discernible objectively as the physical sign and the symptom; the other, the exclusive property of the patient and consisting of modes of sentience, can only be obtained from him; consequently the investigator has to rely wholly upon the good faith of the patient and his descriptive powers. Thus in the matter of a pain, or other abnormal sensation ; its accurate localization ; its character and intensity ; its tendency to be fixed or to wander, whether diffusely or along definite lines; its time relations to certain bodily functions or its independence thereof; its association with a

local tenderness or not (this as elicited by pressure or by directed movements partakes more or less of the nature of the objective symptom) —for all such information we are dependent solely upon the testimony given.

Now, assuming good faith and intelligence on the part of the sufferer, and admitting that the sentient records may be very vivid and precise in all such details, can we rely upon them as positive evidence of the presence of a definite local morbid process of such character and fixity as to justify the diagnosis of organic disease? It is not a question now of distinguishing between organic and functional disorders, as previously defined; it is a question of the possibility that true organic mischief may, or may not, be simulated by subjective symptoms (chiefly subjective, but not wholly unaccompanied at times by objective signs, witness the phantom tumour, though these latter are in general ill defined). Well, experience has taught us that we cannot so confidently rely upon the evidence obtainable, and that the most experienced may be and are at times deceived. Does this mean that the patient has not felt the pain described, for we have postulated good faith? This does not follow, and is not justified by a consideration of all the aspects of the case; for psychologists and physiologists have long recognized a state of the *psyche*, which they have labelled "expectant attention." It is a state which is marked by the direction of the mind upon this or that part of the body with an intentness and a persistence which must be regarded as morbid; it is a state of watchfulness accompanied by apprehension.

It is not necessary to enter into the mode of genesis of this condition of things; whatever the occasion, traceable or not, there the phenomenon is; and it is quite established that during the persistence of "expectant attention," the subjectivity cannot be relied upon as a witness; again, this is not to impugn the veracity of the ego, but simply to recognize that there may be self-deception through the senses, and in the sequence self-delusion through the judgment. The kind of deception most often met with is due to an exaggeration, an exaltation of the bodily sensations, but this is often associated with a distortion or perversion of their character. Upon these false data the apprehensive mind sets to work to construct or rather to imagine the causal complex which is responsible, and, aided by sympathetic friends, the superstructure arises in due time. Need it be said that, the data being fallacious, this superstructure is at best a simulacrum, and as such it confronts both the sufferer and the investigator?

This subject has been touched upon because of its importance alike to him who suffers and to him who is in search of the causes of, and remedies for, the suffering. How many of us are there not, who pass through life the innocent victims of make-believe symptoms, who cannot imagine that feelings so vivid and so well located, one might say so riveted to the part, should own no demonstrable material existence; and how many learned and skilled doctors, in the presence of a suffering so obviously genuine, and so precisely referred to this or that part of the body,

whether upon the surface or deeply placed, are apt to lose sight of the possibility that the semblance of organic disease is before them and not the real thing; whence it comes that all their well-planned and vigorous therapeutic assaults have come to naught—a mere tilting against windmills. It behoves us, therefore, never to lose sight of the state of "expectant attention" as a psychic phenomenon, absolutely real and actively operating, however unsubstantial its demonstrable foundations. We shall be led to suspect the presence of this state the more we become aware that the sufferer is dominated by uncontrolled emotions and that the habit of introspection and self-absorption is in possession; and this suspicion will, in many cases, bring real comfort to the physician when the aspect worn by the symptoms presents the graver features of a serious if not mortal complaint; it would be even more consolatory to the patient if it should prove possible to shake the convictions, so deeply rooted in his sensorial system, that *dis*-ease necessarily signifies disease in the minatory sense which is tormenting him. But how hard to bring this consolation home to those who feel, and who take feeling to be synonymous with those grosser material changes which have dimensions and wear a shape, which they have seen or handled, and in which all their fears are concentrated.

And this state of "expectant attention," this phantom builder, what is it in its essence; where does it house; how does it operate? These are mysteries still to be unravelled.

## CHAPTER VIII

### HEART FAILURE

DEFINITION. When we speak of heart failure, what, precisely, do we mean? We mean that the heart, as a power-chamber, is failing to carry out the purpose for which it was designed and built, a purpose purely mechanical, namely to maintain a normal circulation of the blood through its own compartments and through the associated channels of the vascular circuits. And what do we understand by a "normal circulation"? We understand a circulation which maintains the functions of the body at a level of activity which we recognize as that of health. Needless to say, the normal circulation varies within wide limits, is different for each individual, and varies with the varying circumstances of life, according as this is carried on under conditions of ease or stress. We need not endeavour to define more strictly; we all know what is signified by health.

The heart was designed and built to develop power in the requisite quantity, *quantum sufficiat*; and to bring that power to bear upon the blood contained within its cavities, so as to overcome the inertia of the contained blood mass, as well as that of the blood mass filling the blood vessels, a very much larger quantity; furthermore, so as to overcome the resistance opposed by the

walls of the vessels to the movement of the blood through them. The development of the necessary power by the heart involves, *pari passu*, an accompanying rise in blood pressure within the heart and vessels ; and inasmuch as the resistance of the vascular circuit preponderates so enormously over that of the intra-cardiac passages, so much so indeed that we might regard the task of the heart as consisting almost entirely of the delivery of the blood across the vessels, it follows that as the blood issues from the left and right ventricles and begins the greater and lesser circuits respectively we should look for and find there the maximal records of blood pressure. Thence onwards the blood pressure must fall in exact proportion to the reduction of each circuit, measured from the starting-points at the roots of the aorta and pulmonary artery,[1] until at the points of return, viz. at the mouths of the superior and inferior cavæ and of the pulmonary veins, the journey being then accomplished, the blood pressures would be found to be practically extinguished. The blood pressure is therefore constantly changing as we travel from the heart towards the capillaries and from these back to the heart. The blood pressure which we record clinically represents that prevailing in the medium-sized systemic arteries.

The state of the circulation then gauges heart failure, and we must next ask ourselves in what way circulatory failure may show itself

[1] The word "reduction" here used applies to the reduction of surface friction which no simple linear measurement would give, since the vessels are continuously branching and multiplying their surface areas.

and be brought about. We have seen that the elasticity of the arterial systems (for both the lesser and the greater circuits are concerned) acts the part of a transformer converting the power developed by the ventricular beat into an elastic stretch. This stretch reacting upon the blood contained in the arteries appears as blood pressure, and according as this blood pressure is high or low so is the vigour of the circulation. Circulatory failure will show itself therefore when the blood pressure falls below a certain level, which state must then be labelled hypotension. A considerable range of tension is compatible with health, and for each individual we must interpret the readings of the blood-manometer in terms of the bodily functions; if these correspond with health the blood pressure is normal for that individual. Hypotension may be brought about in two ways: (1) By lack of vigour of the ventricular beat, the extreme case of which will be represented either by a temporary suppression of the beat or by its reduction to a minimum—this is the condition found in fainting; or (2) it may be brought about by a withdrawal of the vascular tone, in particular the arterial tone. The effect of this withdrawal of tone is that the resistance to the heart beat is withdrawn in equal degree, and in consequence the heart, however vigorous its contractions, is unable to beat up a sufficient blood pressure—blood tension being the resultant of the opposition of vessel resistance to ventricular contraction. This state of things obtains in the condition known as collapse or shock.

Another aspect of the state of shock and its

mode of origin may be arrived at by considering the volume of the blood in relation to the capacity of the vascular system. Under normal conditions we must regard this volume as a quantity very nicely calculated to fill the heart and vessels *in a state of normal tonicity*; reduce this tone and at once the slackened grasp of heart and vessels upon their contents makes room for a large addition of blood; and so long as the lowered tone persists it is only upon the condition of the augmentation of the volume that the blood pressure can once more be raised to a better level. Failing this, the ill-filled heart and vessels are unable to get sufficient purchase upon their contents to restore to the heart the driving force, and to the vessels the resistance, essential to an adequate flow of the blood. Of course the recovery of tone reinstates the normal conditions of itself.

If we contrast the circulatory failure of cardiac syncope with that of shock, we note the transitoriness and relative innocuousness of the former (except in rare cases). It is self-evident that the suppression of the heart beats or their depression to a minimum cannot last for more than a very short period without immediately threatening life—the case then becoming a fatal swoon, a rare event. In shock, on the other hand, the condition may persist for comparatively long periods, as we should expect from the sluggish behaviour of tonus, and from the fact that the cardiac beats, though diminished in power, do continue in sufficient force to maintain bare existence. There are thus two forms of circulatory failure marked by hypotonus.

Passing now to the opposite extreme, that of hypertonus, we must put the question—can it be the cause of circulatory failure? And the answer is no. For raised blood pressure is never in excess of the needful, i.e. of the mechanically needful, being always consequent upon other happenings which make their direct call upon the heart for an increased output of force. Thus structural changes in the walls of the arteries which thicken them, and so narrow their lumen, will obstruct the blood flow and call for a more powerful systole; and changes of the same kind will also destroy or reduce the elasticity of the walls, this rigidity imposing upon the heart a greater effort. How this latter factor operates will be most apparent if we consider what would happen if the vascular system were composed of tubes with absolutely rigid walls, for in such case the blood flow could only be maintained in intermittent jets, the whole volume of the blood being propelled forward *en masse* at each systole. The amount of power demanded for this would be enormously multiplied, if indeed materials could be found to bear the strain. Regarded thus, it is evident that any change, however slight, which impairs the elasticity of the arterial walls will tend to raise the demand upon the heart.

The same result will obtain, in the absence of any structural changes in the vessels, from a heightening of the tonus of the smaller arteries, provided this is sufficiently prolonged: in such case the heart is obstructed so long as the tonic contraction lasts.[1] Hypertension is only possible

[1] Every living tissue is possessed of tone, and this will therefore apply to the walls of the capillaries, consequently we cannot

by virtue of the property of compensatory hypertrophy which belongs to all muscular tissue, and when this property comes into play it is to meet and counterbalance adverse influences, which are of the nature of disease. Compensatory hypertrophy being thus a protective mechanism, its operation is always salutary in so far as *the maintenance of the circulation is concerned*, and only when its reserves are exhausted will circulatory failure threaten. This, of course, is not to deny that dangers do attend upon cardiac hypertrophy, but these dangers are of another kind, and are to be regarded rather as accidentals ; they are contingent upon the soundness of the walls of the heart and the vessels, i.e. their ability to stand the strain of the hypertension without stretching or tearing. The dangers of the aneurysm and of rupture constitute an ever-present menace in hypertension, when it exceeds certain limits. Apart from this it appears to be a direct excitant of degenerative (atheromatous) changes in the walls of heart and arteries, and these changes are liable to be followed by a morbid train of consequences, which are started by the tendency of the blood to deposit its fibrin upon the damaged surfaces, whence come embolism and thrombosis. It is these same atheromatous changes which weaken the vessel walls and lead to rupture and aneurysm.

We come back then to hypotension as the immediate cause of circulatory failure, whether

exclude the possibility that the capillaries in certain blood states may contract more or less and be an appreciable cause of obstruction.

the cause be cardiac asystole or hyposystole on the one hand, or vascular hypotonus on the other hand. A third variety of circulatory failure comes now, also accompanied by hypotension, but not necessarily caused either by cardiac contractile deficiency or by lack of vascular tone: it is due to inco-ordinate action of the heart fibres. Whether this form is always accompanied by a diminished contractile force of the individual fibres or not, it is at any rate conceivable that the force of the individual fibres should be unimpaired, and yet the most complete circulatory failure result. Time is of the essence of this form of failure, not energy, for it must be evident that unless the contractions of the individual fibres or groups of fibres be so timed as to co-operate, there must be a thwarting of effective output more or less complete. There has been previous reference to the state of the ventricle of the frog's heart, often witnessed as the result of digitalis poisoning; it bears so strikingly upon the subject under consideration that repetition may be excused. The ventricle is there seen contracting actively, but to no purpose, for the reason that one part is contracting whilst a neighbouring part is relaxing, and this state of things is repeated over the whole ventricular surface; patches of constriction and of whitening, due to expulsion of the blood, adjoin patches of bulging and reddening, due to the filling of the relaxed part with blood. To and fro from part to neighbouring part the blood is driven, but no combined act of propulsion into the vessels is effected. Death in such a case is actually due to asystole, though there are the

fibres contracting before our very eyes, but not consentaneously, and, as the physicist would say, no work can be done by such conflicting contractilities—such is the inco-ordinate heart.

The term auricular fibrillation has been applied to a typical example of this form of derangement, and the term auricular flutter to a kindred condition. Much work has been done in the investigation of these states, and quite recently Dr. Thomas Lewis [1] has published the results of experiments, by himself and by his fellow-workers, designed to throw light upon the intimate nature of the phenomena witnessed. The subject is one of considerable intricacy, and further progress on these lines must be left to the scientific experts who alone are qualified to grapple with the problem. But there is another aspect of things, which, leaving out of account the details of the *anatomical* and *physiological* basis of the manifestations, looks straight at the outcome of the working and considers what is the resultant effect upon the dynamic output of the inco-ordinate heart.[2] Thus considered, how are we to regard auricular fibrillation and flutter? How else than as forms of paralysis, more or less developed, of the chamber affected, or, more briefly stated, as modes of dying, culminating in complete functional death. In its fully developed state fibrillation of the auricle extinguishes completely the auricular systole, and all that remains to the chamber is to make room passively as the

[1] *Ut supra.* Lectures delivered at the Royal College of Physicians, 1921.

[2] Anatomically and physiologically the subject has already been discussed.

blood from the veins flows into it, and to subside as the blood leaves it—the movements of the thin walls following the blood volume as the intravenous pressure determines. We have seen that the ventricle can at need forgo the assistance of the auricular systole and that a working circulation may still obtain, though, no doubt, at a lower functional level and necessitating a restriction of the demands upon the heart. But if the fibrillation extends below the auriculo-ventricular sulcus into the substance of the ventricle, then a much more serious condition arises and heart failure begins in earnest, heart failure from inco-ordination. Now, when we feel the pulse our fingers are upon the ventricle, in so far as we are concerned with its efficiency as a force pump, for we feel the impulse of the systolic wave and we gauge the intravascular tension it maintains, and it is not possible for inco-ordination to proceed to appreciable lengths without the finger detecting this as an irregularity of the pulse, both in rhythm and in force.

Confirmatory evidence can be obtained by means of a pulse tracing, which has the great advantage that it furnishes a permanent record by means of which we can demonstrate to others the inco-ordination our fingers have detected. Given these findings which prove absolutely that the heart beats are disorderly in their sequence and unequal in their power and in their volume-output, does it follow that we are in the presence of heart failure? The answer must be—no, for we do meet with cases occasionally which show inco-ordination of this type, and to which the term delirium cordis is applicable, yet

which do not yield the evidence of circulatory failure; the subjects of this *dis*-order being able to lead active and effective lives unaccompanied by the signs which mark a flagging and a labouring heart. What are these signs which it is incumbent on us to look for in every such case? They are:

(1) Cardiac distress marked by pain of special type and surface distribution; or by that form of discomfort which may attend upon palpitation or upon dilatation of the heart, and though not capable of being labelled "pain" may be hardly less distressing.

(2) Respiratory distress, e.g. oppression, breathlessness; both (1) and (2) are the result of physical effort, in particular.

(3) Undue acceleration of the pulse, quite independently of the irregularity present—this acceleration of the heart, as the fingers record it, must be checked by auscultation of the heart area.

(4) The direct signs of circulatory failure, as shown by:

(*a*) An altered distribution of the blood, whereby the arterial system tends to be depleted, the venous system to be surcharged; the former showing itself in the diminished volume of the pulse, the latter in the visible engorgement of the great venous trunks, and in the signs which indicate a dilated right heart, as well as in the tendency to cyanosis which points to capillary stasis and congestion together with mal-aëration of the blood.

(*b*) Certain sequelæ which depend upon the venous and capillary engorgements, e.g.

serous leakages into the tissues and the serous cavities, hæmorrhagic extravasations and clottings, and certain accidents which may arise therefrom.

(*c*) Functional failure of the organs of the body, respiratory, renal, and alimentary tract (including the liver as an appendage of this tract); together with definite physical signs, especially in the lungs (at the bases), in the kidneys (urinary changes), in the liver (enlargement and maybe pulsation).

In its entirety (4) may be summed up briefly as: the signs and symptoms of "back pressure." In the presence of the signs and symptoms just enumerated, either in whole or in part, we are justified in diagnosing heart failure; but in their absence we can only say that we have no evidence of its existence. True, it may be coming, and even close at hand; but then again it may not, so we must just wait for developments. Any such instance of abnormal rhythm calls for observation, so that at the first suspicion of failure we may take steps to meet the situation; but this observation would be wisely placed in the keeping of those in touch with the case, rather than with the subject of the abnormality, lest the subjectivity become the busy seat of apprehension. Each case, however, must be dealt with, in this respect, on its own merits. Need it be said that, pending conclusion as to the nature of the case, organic or functional, any circumstances or conditions which might tell adversely upon the circulation should be remedied if feasible?

In the end then the evidence of an impaired dynamic output of the heart supplies the crucial test of the presence of heart failure, and to this test every case must be brought.

These remarks hold for heart failure *in esse*, in the aspect; but as has been stated, though not in actual being it may be approaching and imminent, and the question is—have we any means of detecting heart failure *in posse*, in the prospect? We have, but it will not tell us whether it is near at hand or distant; it will only say that it is on the way. What are these means? They belong to our long-established clinical possessions, and consist in the demonstration of enlargement of the heart and in particular the presence of hypertrophy; indeed, if we could dissociate hypertrophy from dilatation and determine in a given case of enlargement how much of this belonged to the one, how much to the other, we should rely solely upon the hypertrophic enlargement, since this is the only evidence we possess of *compensatory* reaction. But though we cannot dissociate these, we can always state that hypertrophy dominates so long as effective circulation is maintained without the presence of any of the above-mentioned signs and symptoms of a flagging heart. The presence of compensatory hypertrophy is proof of an effective cardiac vitality, but at the same time it is proof also that the heart is drawing upon its reserves, and as these have their limitations it is impoverished in proportion to the draught. How much of these safeguarding powers have we used up, in a given case, and how much remains unused? These are questions impossible to

answer at the present time, and therefore we cannot foresee the advent of the coming disaster. We can only say it threatens: it may be postponed for an average lifetime of careless living, *a fortiori* for an unabbreviated term of careful living, and in every case wise restriction of the heart's output will put off the evil day; on the other hand it may be on the threshold and no sign given that the compensatory powers are spent. The insuperable difficulties of prognosis as to length of life reside in this inability to gauge the reserve potentialities. The one practical outcome of these considerations is that whenever we meet with evidence of cardiac hypertrophy we must weigh the question of the cardiac restrictions to be imposed, always remembering that the strictest rest of the body as a whole cannot relieve the heart of the major part of its physical effort, moreover, that the health of muscular tissue depends upon the maintenance of effort, as is seen when we fix a limb in a splint and observe how rapidly the muscle fibres waste. Briefly it may be stated that so much of exercise as can be taken without unduly quickening the heart or shortening the breath may, as a rule, be allowed without endangering life and with advantage to the heart itself; this will apply to bed patients as well as to those who have the more or less free use of their feet.

If modern instrumental methods can furnish us with more accurate means of differentiating between the innocent arrhythmia and the pernicious type, and, further, will enable us to forecast with greater precision, they will be doubly welcome.

# CHAPTER IX

## TREATMENT

## I

*Il cuor fermo—*
The stable heart.

*Vita brevis; ars longa; occasio celeris; experimentum periculosum; judicium difficile. Oportet autem non modo seipsum exhibere quæ oportet facientem, sed etiam ægrum, et præsentes, et externa.*

Life is short; Art is long; Opportunity fugitive; Experiment hazardous; Judgment difficult. Not only therefore must he (the physician) himself do the things which behove the doer; but (he must see to it that) the patient also, and those in attendance, and outside circumstances (co-operate likewise).
HIPPOCRATES, Section I, 1st Aphorism.[1]

DANTE has a passage in the "Inferno" in which he speaks of the firm, or stable, foot (*il piè fermo*); its exact meaning where it occurs has been much debated, but apart from the context none will doubt the value of the stable foot or the need that, in the course of convalescence, the patient should recover this lost possession by every endeavour. If this be true of the foot, how much more does it hold of the central organ of physical life, the heart, upon which, more immediately than any other organ, life depends.

[1] Commentators are in general agreement as to the meaning of the second half of this aphorism, and Littré's French version appears to be a very close rendering of the text; it has been followed almost verbatim in the above.

The stability of the heart is thus all important, and yet no function of the system is more at the mercy of happenings within the world of the Ego and in the world of Circumstance which surrounds it. Not only does it respond to things physical which we can weigh and measure and analyse in the test-tube, but also to things psychical, things intangible, which we cannot so deal with, as when the emotions cluster round it and call the measure of its rhythm. Moreover, no other organ is more liable in its derangement to simulate and pass off its make-believe upon the patient, and, it may be, upon the physician also. This being so, some general considerations may perhaps be allowed before passing on to the discussion of treatment in its main outlines and its one objective, namely to stablish the heart.

And first on the question of Temperament as it bears on the issues of disease and its treatment. The older physicians dealt somewhat heavily in "the Temperaments"; we, in our turn, have become accustomed to pass them by with scant regard; perhaps because the dissecting scalpel and forceps know them not, nor the microscope, as little as the chemist can find them in the laboratory or express them in a formula—is our neglect quite justified? It is not proposed here to enter into the question of the temperaments in detail; it must suffice that we look at the subject broadly, and ask ourselves what we mean by the word temperament and whether or no it stands for something real. The answer must be that it is something very real, something which belongs integrally to the body corporate and completes the make-up of

the whole Man. For what do we see which distinguishes characteristically one individual from another? Let us take an example: we see one type of humanity brimming over with energy, always on the go, enterprising, eager in pursuit, hopefully expectant, almost incapable of seeing the darker aspects of things, incurably optimistic; and contrasting with this we see its counterpart, sluggish, apathetic, lacking incentive, disinclined for any effort physical or mental, distrustful, incapable of seeing the brighter side of things, incorrigibly pessimistic. To these two types, which we may label the sanguine and the ex-sanguine, the rough-and-tumble of circumstances presents itself, including disease—what will be the response individually? Each type will enumerate the same five senses, the same list of organs, and the chemist and the physiologist will be hard pressed to discover any distinctive features in the metabolism and general functioning of the organs of the one or other type; yet how different must we not expect that the reaction will be of these sthenic and asthenic variants, respectively, whose physical output, measured in units of momentum, contrasts so strikingly. On the one hand we may perceive the driving force of a Florence Nightingale, ceaselessly impelling, tireless, it would seem, and often rising superior to severe bodily infirmities; on the other, an organism accepting passively, floating with the stream and, like the stream, seeking out the line of least resistance for which alone its motivity is adequate. How can we explain these different modes of vitality? Perhaps best by likening them to two Leyden jars, as

identical structurally as they can be made, but the one charged with electricity to the limit of its capacity, the other comparatively empty. In such case, whereas the former threatens to become dynamic at any moment across the surrounding resistances, the latter, receptive rather than explosive, stands " at ease " until a further access of energy comes its way. Or we may liken them to two portions of steel from the same casting, and therefore presumably as nearly identical in their structure as is possible. Subject the one to a high degree of magnetization as compared with the other, and we shall witness the same contrast in their relative behaviour: the former, highly polarized, is powerfully attractive or repellent, as the case may be; the latter as markedly distinguished by its feeble tendencies in either direction.

So much for the two types met with in the commonalty of mankind, as considered from the point of view of mere physical vitality—here, the cheerful, optimistic busybody, and there, the dismal, pessimistic exponent of the *laisser faire*. Disease approaching these two will be certain to meet with different powers of resistance, and will shape its course accordingly. But mere vitality, though it must find an outlet, will not necessarily expend itself profitably nor even maximally; to secure this it needs direction from the higher centres of the intelligence, for then purpose comes into view; and purpose gathers intensity of action as the Psyche comes upon the scene and presents its incentives in the form of duty, love, some great desire, personal or altruistic, in one word its Ideals. Let now the

great desire be backed by that tenacity of purpose which we label will-power, and which is a form of courage and of endurance, and we see before us the Man, *justum et tenacem propositi*, just and firmly purposed, whose potency, raised to the *n*th power, is of unimagined effectiveness.

Where does the Psyche reside ? If autogenous but latent, how may it come into active being ? If not autogenous, how may it be implanted ? These are among the chief mysteries of life ; but we must assume, for all history teaches it, that to every human being there belongs, in its own particular form and measure, a spiritual potential which may be elicited or implanted. Assured of this and of the incalculable powers which this spirit force may confer upon the body, it is the part of the physician to secure by every endeavour its co-operation in the campaign against disease, which sooner or later must arise, and which he has to conduct.

This brings us back to our subject, Medicine, and to the first aphorism of Hippocrates, for surely when he framed those inspired words it is unlikely that he did so in ignorance of the psychic factor and its almost miraculous powers to stem the advances of disease, which the occasion manifests. Indeed, are we not justified in attaching to the words of the second half of the aphorism a significance which would point directly to an appreciation of the full value of this factor and of its utilization by the physician ? for after recording in the first place what is his own immediate duty, he adds : " but (he must see to it that) the patient also, as well as those

in attendance and outside circumstances (play their part likewise)." Thus he places first on the list of contributary factors that which the patient can himself supply, and which is obviously something more than mere passivity—to wit, a courageous hopefulness, a determined endeavour to aid and abet as far as in him lies.

The first aphorism meets us at the very portals of the temple of Æsculapius, and like a solemn introit conducts its votaries across the threshold. It tells of the brevity of Life, and of the interminable pursuit of Art (life's task); it tells with unerring directness of the urgency of the Moment, that is, of the swift passing of Opportunity's escaping forelock. The next pronouncement cries halt, despite all the need for haste which the crescendo argument has developed up to this point; for now it bids us pause and consider that Experiment is hazardous, in that, apart from the peril of active intervention, we may perchance thwart the healing powers of Nature; and, again, that Judgment is difficult, not to say fallible. The problem thus confronted, there follows as a corollary the immediate practical deduction which must be acted upon without delay, yet without undue haste, having first given the mind time to visualize the problem in its entirety and to consider the vantage point of Opportunity, in time and place: the second half of the aphorism embodies this conclusion.

Now as to these two very personal possessions, Temperament and the Psyche, i.e. the spiritual potentiality, the former is in no wise at command, either of the patient or the physician; it is part of the original make-up of the individual,

as intimately associated with, or dependent on, the structure of the body, as is the horse-power of an engine upon its build: the aphorism does not envisage this possession. It is otherwise with the psychic factor, for though doubtless as a potential it may be regarded as an original possession, yet in its development for better or worse it may be shaped, adapted, augmented, or suffered to dwindle,—this as the result of individual effort or neglect, for which without doubt the possessor is personally responsible: the aphorism appropriates this property and seeks to utilize it.

Like the *descensus Averni*, the downward path to disease is so facile, whereas the retracing of the steps and the recovery of the upper regions of health so arduous; yet this toil, this task, must be undertaken, and in the endeavour, apart from the labours of the physician, the patient can lend a hand, and likewise those in attendance —the last named in particular as, under the guidance of the physician, they control outside circumstances, *res externas*, and create a harmonious, inspiriting atmosphere.

## II

*Confert an non? Nocet an non?*
Doth it profit or not? Is it harmful or not?

*In perturbationibus alvi et vomitibus sponte evenientibus, si qualia oportet ejici, ejiciantur; confert et facile ferunt (ægrotantes): sin minus, contra. . . .*

In disturbances of the bowels and in vomitings, arising spontaneously, if the matters expelled are such as ought to be got rid of, let them be expelled; it doth advantage and they (the patients) bear it well: if otherwise, the reverse holds. . . .

The above second aphorism of Hippocrates is quoted only in part, but that part is complete

in itself, and the context, whilst amplifying or rather repeating, neither takes from nor adds to the force of the argument as it stands. Looking at the brief pronouncement, we seem to have dropped from a great height, and its commanding outlook, to the low level and narrow limits of a particular symptom and to an argument based thereon, which would appear almost commonplace and self-evident. To repeat it what are we told? That if the matters which are cast out by the vomiting or diarrhœa are such as should be voided, the patient will be all the better for it, and we shall wisely leave Nature alone and not interfere with her cleansing. Why, of course not—a first year's student will be almost indignant at such a platitude. Besides, how are we to know that the things are "such as should be voided"—the argument starts by begging the question in a most flagrant way. Add to this that at the present moment we are supposed to be considering heart disease; what is the bearing on this of vomitings and purgings?

And yet this second aphorism is no unworthy follower of its sublime predecessor, it carries a great argument; for though we have passed from the general to the particular, yet this we must do if we would come to grips with disease, and when we look more closely we shall find that in so doing we have not narrowed the view, for that the implication which the argument contains, though applied here to one limited symptom, holds universally throughout the length and breadth of pathology, and bears therefore directly on heart disease and its treatment, as it does in the case of every other form of malady.

What is this implication ? It is that whensoever we are confronted by the *symptom* we must pause and consider, asking ourselves this question first, whether perchance it be the expression of Nature's own mode of dealing with something noxious which has arisen within the system ? Is this the explanation ; are these symptoms Nature's reaction ; does this reaction tend to re-establish health ? If so, we shall interfere at our own peril. It is not that Nature's reaction always makes for health, nor even then, when that is its direction, that it may not need control of its intensity : it may, but the point is that we must weigh this matter before we take action. Regarded thus we must allow that a very mature mind was needed, when face to face with such violent happenings as vomiting and purging, to excogitate the possibility of the presence of a *vis medicatrix naturæ* at work on behalf of the patient, and to see in these apparently subversive manifestations a possible healthward movement. No less a doctrine than this is contained in the second aphorism, and no less a deduction than the maxim, *primum non nocere.* And this aphorism dates back over two thousand years !

But to repeat, how are we to know that the workings in question are of the nature of a salutary reaction ? We can only do so by observing what the general effect is upon the patient, as a whole ; and here we may have to counterpoise one symptom with another and, summing up the pros and cons, see how the balance stands. In the case of the relief of a sick headache by the act of vomiting, the matter is very simple ; in the case of a uræmia it may be

much more difficult to decide in how far we should seek to control an attack of vomiting and purging. After all, an observant mind and common sense will, as a rule, make it plain whether in the main the patient is benefited or not. Accordingly, the question to be put to the symptom in every case is: *confert an non?* Doth it profit or doth it not? This is the Hippocratic way.

Failing evidence of actual benefit conferred, the next point to be settled, before anything is undertaken, is whether there be evidence of damage inflicted by the symptom, or not; the question now standing, *nocet an non?* Thus we are, let us say, in the presence of an irregular action of the heart from which no obvious advantage derives, but granting this, is it therefore harmful, or is it harmless? If we can assure ourselves of the latter alternative, we may reasonably stay our hand and be content to watch the course of the arrhythmia. This again is the Hippocratic way, the essence of which is that the symptom is always to be referred back to the patient and tested as to its significance by the way in which it is borne. Hippocrates looks at the symptom, yes, but then he turns and looks at the vital momentum of the whole organism, and he finds in its persisting strength or in its impairment the true measure of the symptom and the therapeutic guidance which he seeks.

Such is the order of interrogation of the symptom. Let us now pass on to consider the interrogation of the *remedy*, which after due deliberation has been selected, be this remedy

operative or medicinal; the questions asked are the same, but the order is now reversed. We now put first the question, *nocet an non?* having in mind the possibility that in some degree, greater or less, we may hinder Nature's efforts to overcome the disturbance. Here we shall remember that the main action of a remedy may be in the right direction whilst some by-effect may thwart the intention; and we may recall that in the older prescription this was kept in mind by the injunction that the aim of the medicine administered was to operate quickly, safely, and as pleasantly as might be, *cito, tuto ac jucunde*: it is the "safely" to which we now refer. Having satisfied ourselves on this point, our next question is, *confert an non?* i.e. does the remedy render positive assistance to Nature, or not? In this way the *primum non nocere* is supplemented by the *secundo prodesse*, in which two maxims the aim and end of all treatment will be found, and virtually these two are contained in the second aphorism.

To proceed: in estimating the significance of the symptom and the value of the remedy, we gauge the one and the other by the reaction of the whole system to the morbid factor on the one hand, to the medicament on the other. This reaction consists of effects which are general, i.e. which the system, as a whole, manifests, and effects which are particular, which belong, that is, to a special organ or function. The former may be striking, but often the general effects are inconspicuous and require a trained observation to seize them and their import. A sense of well-

being, of the presence of reserves of vitality, may be manifested in many ways, very tell-tale in the aggregate, but, severally, to be sought and found rather as hints or suggestions which show but faintly on the surface. But despite their vagueness these may mean much, indeed much more than that which the particular organ or function may display unmistakably, even noisily, as—let us say—in the case of a loud valvular bruit at the heart. " I like the looks of you," addressed to the patient, was perhaps the chief contribution of an experienced and wise physician, at a bedside consultation in a case of prolonged and serious illness. The homeliness of the phrase carried its own comfort, and was perhaps chosen for that very purpose ; it certainly put a new heart into the patient as well as into those who ministered to him, and thus brought the whole household *et ægrum, et præsentes* into the fighting line with renewed ardour : the patient recovered—*post* or *propter* ?

Accordingly, in summing up the points of the " case " with a view to forming a judgment upon the present state—a forecast as to the future—and the line of treatment to be adopted, the general impressions gathered, however vague, may not only not be ignored, but must be allowed a prominent place, and we must be prepared on occasion to give them precedence, and maybe a determining voice in the final decision.

A word about the instrumental record. In the days before we possessed the thermometer, the physician thought of " fever " and measured it in terms of bodily reactions ; by fever I mean the

pyrexial infection, of which raised temperature is an essential feature. Thus the hot surface of the body, the dry skin (in general), the flushed face, the headache, the tendency to mental confusion, wandering, or even delirium, the quickened pulse, the scanty, high-coloured urine, the furred tongue, anorexia, and constipation (in general), etc.; these constituted so many criteria whereon he formed his conclusions as to the severity of the attack. Nowadays we are apt to let the record in degrees of quicksilver measure the fever for us, and to pay less attention to reactive evidences. Of course one does not want to exaggerate, and none would desire to revert to the pre-thermometer days, for the value of the recorded fact is incalculable; but still there is the tendency to let the thermometer record come between us and the patient, instead of making it the starting-point of all those other investigations into the bodily reactions, which the elder physician so patiently pursued; which reactions indicated the individual tolerance of a given rise of temperature by an individual person, and showed us not an abstract case of fever but a concrete feverish patient,—a patient, i.e. a man suffering under the incidence of disease, and bearing it, how?

The same comment applies to all other methods of instrumental record; we must accept them, and gratefully, but we must never let them intervene and separate us from the malady as it lives and moves and has its being incorporate in the patient. Always we must bring back the record to the patient, and criticize it in the light of the bodily reactions.

One other point. Each instrument has its own life, so to speak, and tends to go its own way according to the laws of physics which govern it. Not always does the instrument conform itself strictly to following the corporeal movements it is intended to record, and by as much as it does this it fails to be a true copy. Thus the lever, which receives its jerk from the spring pressing upon the artery, tends at a certain point to continue its direction after the spring has begun to descend with the subsiding pulse; in so doing it leaves the spring for a brief moment, and the curve it traces during that moment does not record the arterial movement. Why does it do this? Because it is bound so to do by the law of inertia which governs its construction. We must therefore always bear in mind the fallacy of the instrumental error, which the mechanical recording appliance may introduce. Are we then to abandon such mechanical appliances? Certainly not; all that we have to do is to remember their limitations.

Lastly, when the *present state* of the patient has been completed, and the whole disorder, analysed into its several parts, lies before us, we shall have to remember that the different organs have different values, according to the importance of their functioning, in the economy of the system at large; and that they will thus take rank as primary, secondary, tertiary organs, or functions, and so on. Now it may well be that an organ of low rank may yield symptoms which superficially regarded will be much more striking than the symptoms manifested by a primary organ;

of this we must beware, lest we present our case with the accent in the wrong place. It will not be necessary to elaborate this caution, but as an instance of where the stress should be laid it may be stated here that of all organs in the body the most central, the most fundamental, the organ upon which systemic life depends more immediately than upon any other, is the circulatory; and therefore that in all the graver forms of illness the behaviour of this organ must be watched before all others. So long as this organ gives evidence that it has good store of powers in reserve, we may feel comparatively reassured as to the absence of immediate danger.

## III

τὰ δέοντα,

(*scilicet*) *quæ oportet facientem seipsum exhibere.*

The things essential,
(to wit) those which it behoves the doer (the physician) himself to perform.

The whole of medical practice lies here, in these few words taken from the first aphorism, as already quoted.

What, then, are these essential services which it behoves the physician to render? In the first place he must determine the *present state* of the patient, and this determination demands not only that he should ascertain as precisely as possible how the patient stands actually, at the moment, but he must also endeavour to find out how that *present state* has been reached; in other words, the steps which have led up to the stage of the malady which is before him; whether,

for instance, the development has been sudden or gradual, by fits and starts, or by an even progression. In this way he is enabled to view the morbid process as a living, moving entity, and he acquires a notion of its momentum at the same time that he gets some idea of the probable rate of progress: upon this, with all caution, he may adventure a guarded forecast. The *present state*, then, must include the state of the tissues and organs of the whole body at any given stage of the illness, for from this state we may gather both the force of the morbid process and the resistance of the tissues to its incidence; besides this it must include an idea of the rate of movement of the process, in order that we may picture the situation to ourselves as it really is, viz. a *status movendi*. This does not exhaust the demands which the *present state* makes—it must also include, if possible, a knowledge of the nature of the morbid factor, the *materies morbi*, which first started the disorder, for much information has been stored by this time of the modes of action of the different factors which are responsible for disease; and in some we recognize well-defined affinities and vulnerabilities; and in all we suspect that such exist, as we hope, in time, to discover them. Thus in the group of the acute rheumatic diseases we recognize the presence of a morbid element very vulnerable to certain medicaments; the like holds for the malarial affections; the specific forms of other diseases show the same kind of thing, and so on. This knowledge acquired, the particular disorder before us presents not only a morbid momentum, but a specific morbid

momentum in which we deal with quality as well as quantity, and in accordance with which our treatment must be both qualified and quantified.

Thus, the first thing required of the physician, as a preliminary to treatment, is investigation, the most complete obtainable, with a view to the determination (1) of the state of the tissues and organs of the patient; (2) of the strength of the morbid movement, as judged by the history of the case; (3) of the specific nature of the morbific factor or factors.

But the leisurely procedure which such careful diagnosis demands is not always provided. At times disease sets in with such terrible rapidity that, almost before we have had time to realize the situation, death is on the threshold, *instans tyrannus*. A glance at the sufferer in rapid survey, a quick finger upon the pulse, may be all that is permitted. Then it is that the significance of the *occasio celeris* of Hippocrates is borne in upon us—the swift escape of opportunity. Instances of such happenings are frequent; we meet with them in all degrees of severity, in states of shock or collapse, however brought about, whether, for instance, by intensity of pain, as in angina pectoris; or by severe hæmorrhage; or as a consequence, say, of perforation of the alimentary tract into the peritoneal sac; or, again, by the entry into the blood of some virulent poison, e.g. snake poison. In cases such as these, there is time only to recognize the immediate threat to life and to note at what point the threat converges. Here it behoves us to have in mind the two functions upon which,

more immediately than any other, systemic life rests, namely the respiratory and the circulatory; in the default of the one or other of these, life is most quickly extinguished. Of the two, the circulatory function takes unquestionable precedence; and within its compass, which comprises cardiomotor and vasomotor activities, it is upon the former that life pivots more essentially and more instantly.

The diagnosis in these cases of emergency must be followed at once by treatment directed to the maintenance of the breathing and the circulation; the stimulant being introduced by inhalation, or by way of the alimentary tract (*per os* or *per rectum*), or hypodermically, whether subcutaneously or intravenously. The diffusible stimulants ammonia, alcohol, ether are thus employed, and of these reagents, ammonia and its preparations are, perhaps, less commonly used than they should be; we allude in particular to the use of the strong solution of Ammonia, B.P., introduced into the nostril by means of a feather.[1] In this action the powerful local stimulation of the nervous expansion in the nasal mucous membrane is without doubt the chief reviving factor, and not that stimulation which follows upon absorption into the blood. Where we desire the latter effect, ammonia will be best administered by intravenous injection according to Professor Halford's recommendation in snake-bite (12 minims of the Liq. Ammon. fortis, diluted with three times its volume of water;

[1] In a case of collapse in which the respirations had wholly ceased, in consequence of a poisonous dose of carbolic acid solution, this means proved successful when all others had failed.

the ordinary Liq. Ammoniæ, B.P., is a little less than one-third the strength of the Liq. Ammon. fortis).

Other remedies, called for in similar emergencies, are strychnine salts, caffeine, in the form of the sodio-salicylate or sodio-benzoate preparations, and camphor in sterilized oil. These remedies, if less rapid in their stimulant action, are less fugitive, and they are invaluable. Strychnine must be given in large dose if it is sought to prove its value, not less than gr. $\frac{1}{20}$ to $\frac{1}{10}$ to meet the critical stages; the former dose, gr. $\frac{1}{20}$, may be repeated every six or four hours under watchful observation, but to meet the urgency of the moment it is best to give gr. $\frac{1}{10}$ as the initial dose of the adult. On the first appearance of any suspicion of overaction the dosage can be withheld, the strychnine being replaced by the caffeine salts above mentioned, in doses of gr. iij or iv at like intervals. Which of these two remedies is the more valuable, administered as stated, is difficult to determine, but in grave states of heart failure in which it is desired to allow a longer interval before the next injection, say for the sake of sleep and therefore at bedtime, the writer's practice has been to give strychnine gr. $\frac{1}{10}$ the last thing at night, associated with morphia gr. $\frac{1}{6}$ or $\frac{1}{4}$. The doubt as to which of the two, strychnine or caffeine, should be preferred, makes it advisable, where repeated injections are necessary, to give them alternately, e.g. strychnine gr. $\frac{1}{20}$ and caffeine gr. iij, one or other, every three or four hours. In this way the stimulation would be maintained with much less likelihood of the development of strychnine

toxic symptoms. Camphor dissolved in sterilized oil may be given as an alternative to either strychnine or caffeine in doses of gr. iij (in milder forms of circulatory failure gr. iss may suffice). This dosage may be repeated as in the case of the other two remedies. Its stimulating effect is often experienced earlier by the patient, but owing to the volatile nature of camphor it is probable that the effect is more passing. In this respect it would approach rather to the diffusible stimulants. It is of great value as a circulatory stimulant.

Morphia and opium occupy a place by themselves; they are distinctly tonic to the heart, and unsurpassed by any remedy in the treatment of the severest forms of heart failure, with orthopnœa, dropsy, and sleeplessness. The hypodermic of morphia or the opiate draught may nearly always be associated with the use of the strychnine, caffeine, and camphor group, and where pain and sleeplessness are prominent this combination must always be considered.

But though in circulatory failure we must always think first in terms of the heart, in many cases the part played by vascular atony is scarcely less in evidence. We must be prepared therefore to supplement the cardiac stimulant by the vascular stimulant, and what we mean thereby essentially is the arteriole stimulant. For it is obvious that in the presence of the low blood pressures which mark the state of collapse, it is not much use to whip up the heart to increased effort, unless an increased arterial tone supply the necessary counter-resistance to the escape of the blood from the arteries, and so

enable the ventricles to beat up the required blood pressure. In the state of collapse the relaxed arterioles are the equivalent of a leakage in the high-pressure system of the steam engine; the power-chamber in such circumstances cannot develop its power, and thus it is in the case of atony of the arterioles.

The kind of remedy here demanded is represented by pituitrin, in dose of $\frac{1}{2}$ to 1 c.c., 8 to 16 minims, or by eserine, in dose of gr. $\frac{1}{120}$ to gr. $\frac{1}{100}$ up to gr. $\frac{1}{50}$. Either should be associated with cardiac stimulation whenever we feel that ventricular weakness is present along with vascular atony. When thus combined, the use of the pituitrin or eserine should accompany or follow *directly* upon the use of the cardiac stimulant. If time allows, i.e. if the heart failure does not threaten too urgently, it would seem a better economy of the vital forces to let the vascular tonifiant precede the heart stimulant; this latter statement, perhaps, is on theoretical grounds rather than upon actual observation, but quite clearly it is reasonable, *a priori*, that the increased force of the heart's contractions would be more profitably spent if the desired tone of the arteries were first established.

Another question arises here, not touched upon in the foregoing anatomical and physiological considerations, namely that the bulk of the blood, its volume, plays of necessity an important part in the physics of the circulation; for just as in cases of vasomotor paralysis the vascular system becomes, as it were, too large for its contained blood, so that it is impossible

for the heart to keep itself distended and thus maintain the required blood pressure; so in the case of a severe hæmorrhage the vessels become so much depleted that again their capacity is too large for the volume of blood that remains over, and again it is impossible for the heart to maintain an adequate blood pressure. In the circumstances of collapse brought about by hæmorrhage, the state is further aggravated, because vascular atony adds itself to the emptied condition of the vessels, and for a double reason the blood pressure fails. In this predicament cardiac stimulation, though supplemented by vasomotor stimulation, may not suffice, and it may be necessary to have recourse to another form of treatment which in a sense is purely mechanical, viz. the injection into the vessels of a volume of blood sufficient to meet the deficit.

To this end we may transfuse directly from one person to another; or we may inject saline solutions into the subcutaneous system, in large volume, relegating to the lymphatic channels the task of replenishing the blood vessels, or, more immediately, we may inject these same solutions straight into the veins. It is, however, one thing to pour fluid into the vessels, another to keep it there; the leakage by diffusion (exosmosis) may soon reduce the blood to its former volume. The quality of the fluid used for the above purposes is therefore of great importance, and it is here that the employment of solutions of gum acacia, as advocated by Professor Bayliss,[1] marks a great

[1] Intravenous injection in wound shock—Abstracts of Oliver Sharpey Lectures delivered before the Royal College of Physicians, April 30 and May 2, 1918. Professor Bayliss shows here

advance in this method of treatment, the bland character of the fluid and its low diffusibility constituting the chief qualifications, inasmuch as its use is mainly or largely for the volume it provides.

The ideal use of these gum acacia injections is in cases of severe hæmorrhage, but, quite apart from loss of blood, they may be indicated in any or every state of profound collapse or shock,[1] when low blood pressure is a prominent symptom and the combined use of the cardiac and vaso-motor stimulant has not sufficed—certainly therefore they must always be had in mind.

Thus much in barest outline, for no more is intended, concerning the measures to be taken and the lines of procedure, in the graver and more acute forms of circulatory failure, which constitute the emergency type.

Let us pass to the consideration of the less acute forms of heart failure, which again we can

that saline injections are of merely "temporary value, and liable to be followed by a greater fall of blood pressure"; that "a colloid must be available to remedy the defects of such solutions." He shows further that starch, dextrin, and sugar will not serve, and that proteins are "inadmissible on account of anaphylaxis"; that gelatin and gum arabic both answer the requirements in respect of viscosity and osmotic pressure, but that there are "objections to gelatin on account of its liability to contain tetanus spores," and, more serious still, because it may "cause intravascular clotting, as found by Dale and Richards." He advocates the use of a 6 per cent. solution of the gum, which strength has been found to be quite innocuous "even in a volume" equal to one-half of the total blood volume." The blood pressure is maintained indefinitely by such solution, which produces no hæmolysis or agglutination, whilst it appears to be a good metabolic medium as favouring oxidation processes (from the *Brit. Med. Journal*, May 18, 1918).

[1] Cf. experiences of Captains Cowell, Drummond, and Taylor referred to by Bayliss.

only touch upon in their essentials, and even these we must treat very broadly and generally. At the outset be it repeated that heart disease *per se* does not concern us, as such we do not treat it, but only to the extent that it shows us heart failure in actual being or in prospect, whether in the near or far approach.

If in doubt as to the presence of heart failure *in esse* or *in posse*, prudence will bid us adopt prophylactic measures to the extent we deem necessary, in order to be on the safe side: this we shall carry through, for a while at any rate, under observation, until satisfied one way or the other. The presence of enlargement, if we can make sure of it—a matter often of great difficulty, especially in roomy and emphysematous chests—must always be taken as indicative of heart strain; for even though we may have reasonable grounds for regarding the enlargement as hypertrophic in the main, and are assured that the circulation of the blood leaves nothing to be desired—in other words, that compensation is complete—yet the mere fact of hypertrophy is proof that the heart has begun to fall back upon its reserves, and that, failing due economy, disaster lies ahead.

And now to endeavour to look at the treatment of heart failure in its general aspects, and to ask ourselves what it is we aim at achieving; what the means at our disposal, and how we are to bring them to bear upon the circulatory apparatus.

To the first question—what is our objective?—the answer is: To maintain a circulation as

efficient as possible; for as long as possible; and at the least cost to the patient, i.e. with the minimum of restriction upon, or interference with, the general activities of the body. It is imperative at the outset that the patient recognize that a price has to be paid to secure these benefits, and that whilst he may rightfully demand of the doctor that the restrictions and irksomeness of the treatment be reduced to the lowest limits, this cannot be done without his willing co-operation in accepting the inevitable minimum. The maximum of effectiveness is available upon these terms only.

Such being the problem and its conditions, what are the structures to which the therapeutic appeal is to be made?

(1) To the muscular fibre of the heart as endowed with contractility. The propulsive force of the heart depends upon this property, which by medical treatment may be augmented or diminished.

(2) To the same muscular fibre as endowed with tone. Tone is a state, whereas contractility refers to an act; tone is static, contractility dynamic; tone is, as it were, a taking off ground whence contractility develops its power. These statements are relative rather than absolute, for variations in tone are accompanied by movement, but the movement is of so sluggish a character that for practical purposes it has no propulsive value. Tone is of great importance as regulating the size of the chambers of the heart, and in this way conditioning appreciably the task which contractility has to perform. Thus, if tone be diminished the heart chambers become

dilated, and the propulsive act has to be exerted upon a larger mass of blood, therefore, *cæteris paribus*, must be proportionately more forceful, and *vice versa*. Between the extreme limits of tone in either direction lies a middle term which we recognize as normal tone.[1] Tone may be augmented or diminished.

(3) To a co-ordinating apparatus, which may be styled intrinsic, because imbedded in the walls of the heart, by means of which the contractions of the systems of muscle fibres composing the heart muscle are brought into harmonious co-operation, to the achievement of a purposeful act, namely the circulation of the blood through the heart chambers and through the larger and smaller vascular circuits. This is the sino-auriculo-ventricular nodal system; it is responsible for the automatism of the heart, and in respect of its functions, as initiating, distributing, and co-ordinating (in their distribution) the stimuli which set free the energies latent in the muscle fibres, it is a nervous apparatus. What does this co-ordinating function show us? It shows us rhythm, i.e. an orderly sequence of events, in which the auricular contraction precedes the ventricular, and then a pause ensues; this sequence of three constitutes the cardiac cycle. Further, it shows us this cardiac cycle repeated indefinitely in close and even succession; the evenness applying not only to the relation between the events within each cardiac

[1] It should be noted that the tone of the heart and the tone of the vessels (the arterioles in particular) must be in counterpoise; there must be some sort of balance or proportion between these two.

cycle, but also to the relation between successive cardiac cycles. Moreover, the evenness applies as much to the spacing of the acts in time, as also to the magnitude of the acts themselves, i.e. the pauses between the systoles are equal, each to each, and likewise the magnitudes of the systoles. This statement holds certainly for the left ventricle.

Now, just as the amount of energy developed from the muscular fibre may be augmented or decreased by treatment, so the stimuli which set free the energy from the fibre, and which themselves are generated within the co-ordinating apparatus, may themselves be augmented or decreased. The contractions of the heart may therefore be controlled as to their force *via* this intrinsic nervous apparatus. But besides this, in cases where disease has impaired the co-ordinating powers of the sino-auriculo-ventricular nodal system, and irregularities have appeared, these irregularities both in time and in force may be corrected by treatment, and out of arrhythmia rhythmia may thus re-emerge.

(4) To a co-ordinating apparatus which brings the heart as a whole, in the completeness of its automatism, into close touch with the numerous vascular areas, which severally correspond to the organs and tissues of the body corporate. As the needs of these individual areas, *in combination*, influence the whole state of the body and make their resultant demand, so by means of this co-ordinating apparatus the beat of the heart is brought into harmonious relation with the general need and is adapted in its force, in its volume, in its rate, and in its regularity of

sequence. In so far, however, as the needs of the individual parts, *severally*, are concerned, i.e. as separate entities, these have to be met by local modifications in the vascular tone of the individual parts, whereby their irrigation is made more or less free.

This latter co-ordinating apparatus may be styled extrinsic, because external to the heart area; it is situated in the central nervous system, and consists of nerve centres and nerve tracts, in close functional association with the sympathetic system; the latter, as a structural annexe, serves a decentralizing purpose. The whole is eminently susceptible to the influence of medicaments, the virtues of which may by its means be reflected and projected either upon the heart or upon the vessels or upon both these parts of the circulatory organ. It may, however, prove a matter of great difficulty, in a given case, to disentangle the effect of the drug as it falls upon the intrinsic automatism of the heart or upon the extrinsic governance of heart and vessels, which the cerebro-spinal and sympathetic systems provide. It may indeed be impossible to differentiate between these two, and we must then be content to record the general outcome of the treatment, awaiting further knowledge for the analysis of the complex effect.

The therapeutic appeal under this heading is mainly to the great centres in the medulla oblongata and upper part of the spinal cord, which preside respectively over the cardiac and vasomotor functioning. *Via* the cardiac centres the heart's action may be increased or diminished in frequency, and along with these effects provision

is made that as the frequency rises the force of the beat falls, and consequently the blood pressure; and *vice versa*, with the reduction of the rate the beat is strengthened and the blood pressure raised. *Via* the vasomotor centre the vascular tone, in particular the arterial tone, is largely controlled; and we have seen what an all-important factor this is in determining the response of the heart.

(5) There remains the therapeutic appeal to the blood itself. The blood is the projectile, the heart the projector, and we have seen when discussing the subject of the volume of the blood, what an essential factor mere mass is in a problem so mechanical as that presented by the circulation. We need not enlarge further upon this, but must amplify and complete things by a reference to the part played by the nature of the fluid. A little reflection will make evident how important this question is; for if we think of the fineness of the capillaries, in which the heart meets with its main obstruction, and imagine the difference in resistance which the walls of the capillaries would oppose respectively to, say, Ringer's fluid on the one hand and on the other hand to a solution of gum acacia—we shall recognize how different are the two problems. Each of these fluids would be almost equally indifferent to the tissues, but what a difference in their coefficients of friction. The reference here is to a solution of gum acacia of 6 per cent. strength, of which Professor Bayliss informs us that it may be added, with perfect innocuousness, in the bulk of one-half the total blood mass, that is to say that the total content of gum acacia

in the mixed blood and gum solution would be equal to 2 per cent.—a very appreciable quantity. Have we not in these facts the explanation of the maintenance of the blood pressure by such solutions of gum acacia ? for the low diffusibility which characterizes them will maintain the fullness of the blood circuit, and the viscosity which also marks them will so raise the capillary resistance that the effort demanded of the heart will also be maintained. Of course this effort-demand will exact the means to keep the heart substance amply supplied with nutriment of suitable assimilability [1]—this also will lie among *τὰ δέοντα*, i.e. the essential things required of the practising physician.

This must suffice the present purpose of the writer. We have just considered under five headings the structural parts to which the therapeutic appeal may be made. What then are the means at our disposal to answer the appeal ? They belong to the order of the medicamenta which in their wide range constitute the materia medica as embodied in the pharmacopœias of the world. To those likely to be selected cursory reference has been made, when dealing with the treatment of the circulatory emergency, but this list will require almost indefinite expansion when we come to consider heart failure in all its varieties and degrees, and especially under the headings (4) and (5). The therapeutic field covered

[1] Be it recalled that this gum acacia solution appears to favour oxidation processes, also that, as Professor Bayliss argues, the gum, chemically, is not un-akin to certain carbohydrates natural to the blood.

by these headings is, however, far too extensive to be dealt with here; but heading (3) as introducing the problem of the inco-ordinate heart may be touched upon in respect of one of its forms, which is as striking in its characteristic symptoms as it is in its responsiveness to appropriate treatment.

The class of remedy suitable to the inco-ordinate heart, as exemplified by the rheumatic heart, which is accompanied by mitral valve disease and has developed the characteristic pulse, already described, together with marked signs of circulatory failure, is typified by digitalis. This medicine belongs to a group which includes squill, and probably would include barium salts were it not that certain by-effects of these latter have disqualified them thus far; but, as it stands, digitalis so dominates its group that, in effect, it stands alone. Digitalis is somewhat slow in developing its action, with ordinary dosage *per os*, taking some two or three days; but by hypodermic medication this time may be shortened. Still, even when thus administered it will not compete in celerity of action with caffeine, strychnine, and camphor, or with ether and ammonia, used similarly, in the treatment of the acuter and more intense forms of ventricular asystole, and of cardiac and vascular atonia. Given, however, its own type of case, it stands *facile princeps* among remedies, though it may with great advantage be associated with the drugs just mentioned, and also with opium preparations; but whilst these adjuncts round and complete a harmonious co-operation, it is digitalis which strikes the keynote.

We know, as the result of the labours of recent years, that the typical digitalis-heart case is that in which the auricle is found to be in fibrillation; the knowledge is of great value, and will almost certainly lead on to further advances in pathology and medicine, but it cannot be said that we have had to await the discovery to learn the indications for the use of digitalis, and the rules governing its administration. These had been very clearly laid down long previously by the clinicians and very effectively put into practice; and the writer recalls well, even in his student days at University College, how Dr. Ringer used to teach that if one were called upon to prove the value of drug treatment to the medical sceptic, no more striking example could be chosen than that of the use of digitalis in the case of the mitral valve lesion with water-logged tissues (the result of the circulatory failure) and the characteristic irregularity of the pulse in force and rhythm; and he would lay stress on the fact that it was in proportion as this type of irregularity was present that the efficacy of digitalis would declare itself. How conscientiously and ably this subject has been handled is plain to see in the pages of Dr. Balfour's treatise,[1] in which he enters into most careful detail in respect of the administration of digitalis.

With this brief reference to the inco-ordinate heart, one form of which has alone been touched upon, we must conclude our subject, for, as stated, the headings (4) and (5) of the therapeutic appeal would lead us too far and could not

[1] *Clinical Lectures on Diseases of the Heart and Aorta.* 2nd ed., 1882. Lecture XIV.

come within the scope of this "contribution to cardiology," which makes no pretence at being a systematic work on the Science and Art of Medicine.

Indeed the real objective sought will have been attained if, apart from any value which may attach to the special views on various points put forward here and there in these pages, the anatomist and pathologist may be led, in the presence of the statics of death, to endeavour to reconstruct the dynamics of Life; and the physician be led in like manner, in the presence of the disturbed dynamics of Disease, to recall the teachings of physics and chemistry and physiology, and to endeavour to enlist them on behalf of the healing art. For it must be insisted that disease, whilst defacing, does not efface the features of health, which can still be traced throughout the gravest maladies; and it is from the persistence of this likeness that we infer the persistence of a healthward tendency, that will surely reassert itself, given the opportunity. Upon the presence of this tendency we base all our efforts to recover in their purity of outline the lineaments of a restored health. This likeness, this tendency to revert, is the *vis medicatrix naturæ*.

### *Finalia*

There remain a few words to be said by way of completion, but they need a text whereon to build the argument. I find this text in the arms of the Royal College of Physicians of London, with its subjoined motto. On the title-page of the present treatise is figured a diagram

of the heart with its main vessels, as the central feature of the circulation, but, as shown, it is a mechanism at a standstill, and such as we see it after death. It is not inappropriate, therefore, at the close of a book, the aim of which throughout has been to see structure always in action, to end with a figure which symbolizes the very act which the heart was designed to perform, namely to maintain a flow of blood through the vessels. The arms show "a hand extending from the clouds grasping the arm of a sick man, with half fleur-de-lys in the edge of the field, and in the base a pomegranate as shown in the letters testimonial." The subjoined motto consists of the opening words of the first aphorism of Hippocrates. The grant of the arms was in 1546.[1]

To take the motto first—nobler words could not have been selected: they point at one and the same time to the brevity of Life and the illimitable length of Art—life's task. In them lies the argument for the existence of the physician; here is his part in the scheme of things, namely to conserve life, or even to extend its natural term, if this be possible, and to the best of his ability to lighten the load of disabilities which are wont to cumber life; this last portion of his task is tantamount to a prolongation of life. And why all this—why? In order that Man, within the short compass of the time allotted to him, may achieve as much as possible of Art, the illimitable. We are in the presence of a mystery for which mankind feels that an

[1] For the authority to use the arms of the college I am indebted to the President of the College, Sir Norman Moore, Bart., M.D., LL.D.

explanation is owed, and which some feel will be forthcoming; but, whether or no, doubt there is none in the mind of the physician as to the obligation which is laid upon him.

To come next to the figure in the centre of the heraldic field—the hand grasping the sick man's wrist: What does this typify? That the most fundamental function of the body, that which is amongst the very earliest to take shape in the course of development, and that upon which systemic life pivots most immediately, is the circulatory function. For the hand which grasps is the hand of the physician, and that which is grasped is the artery of the patient, and that which is felt is the beat of the heart as it pulses beneath the palpating fingers. Though we could put our hands directly upon the living heart, we could not gauge so accurately the efficiency of its working as we can by the palpation of the pulse; for it is by the qualities of the pulse which the heart develops that we can best determine the effective value of the circulation. We are reminded then by this figure that the foremost duty of the physician is to feel the pulse; and as it is his first duty, so likewise it will be his last duty as the term of life draws to a close. How long can the patient live?—it is a frequent question, often unanswerable; but so long as the pulsating thread of blood can be detected beneath the fingers, the physician, recalling Galileo's memorable words—"E pur si muove"—can say, It yet doth move, Life is still with us and perhaps all life's possibilities.

And the half fleur-de-lys—What of them? Our President, Sir Norman Moore, tells me they

" are introduced as marks of personal service to the Crown." As such they cannot be said to be of Hippocratic significance, i.e. to concern us as practising physicians, and we must pass on to the pomegranate and put the question: Why this fruit? Again I am indebted to Sir Norman Moore, who tells me of its " high mediæval reputation as a remedy for fever." As far as I can gather this reputation held rather for the East than for Europe, and it has not maintained itself; in other respects it would appear that the pomegranate has played a very subordinate part among the medicamenta. Sir Norman hints that it may have been introduced for other reasons, and perhaps have had reference to " Catherine of Aragon, who bore it in her arms." If so, it, and the tragedy of that queen which it would memorialize, would belong to history, but scarcely to us as doctors.[1]

But why was it not the poppy head? On a first glance I had mistaken it for this, and, knowing that heraldic representations were not always exactly life-like, had eagerly seized upon this interpretation. The disappointment was correspondingly great when I was undeceived. Why was it not the capsule of Papaver Somniferum; source of opium; premier among remedies; *the juice* par excellence of our pharmacopœias? For then should have been chosen a worthy representative of the potencies, which the healing virtues residing in plant and mineral place at the disposal of man. An old physician has said: " Without the poppy, and without the prepara-

[1] I am reminded here by a critic of its use by the early Italian painters as a symbol of suffering.

tions made therefrom, medicine would be maimed and halt" (*sine papaveribus, et sine medicamentis ex eis confectis, manca et clauda esset medicina*). It is so; and it is none the less so because the great virtues of opium may be abused and may then become a terrible scourge.

In the absence of the poppy capsule from the arms of the College of Physicians, may we not utilize the presence of the pomegranate, in its superficial resemblance to the former, to recall *the great remedy* and its congeners, and remind us that it will not be for want of effective weapons in our therapeutic armoury if we fail in our efforts to bring relief?

EXPLICIT

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